THE EFFECTS AND CONTROL OF DISEASES ASSOCIATED WITH EXPOSURE TO ASBESTOS IN A NAVAL DOCKYARD

by

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ABSTRACT

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A review of the literature relating to the diseases associated with exposure to asbestos is followed by a description of processes involving materials containing asbestos, and the men employed, in HM Dockyard, Devonport. The development of preventive measures is explained, and details are given of the precautionary methods which have been introduced as a direct result of the present survey.

An account is presented of an extensive survey of the dust concentrations occurring in most of the processes involving asbestos materials in the Dockyard. This survey shows the degree of risk to which men have probably been exposed in the past, and explains how many men, not previously thought to have been at risk, have been exposed to high dust concentrations.

A detailed study of the clinical, radiological and physiological changes occurring in the men exposed to asbestos is described, and this is followed by a proportional mortality study of the lung and gastro-intestinal neoplasms occurring in Plymouth males.

The report concludes with a general discussion of the data.

INTRODUCTION

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In March 1965 twelve men of the 120 working as laggers in Devonport Dockyard were discovered to have basal rales at the time of their routine Annual Medical examination. These were dry, crackling rales most easily heard over the lower anterior chest and in the axillae. Apart from occasional dyspnoea the men had no complaints and there was no obvious cause for these sounds except that all the men had been exposed to asbestos dust. The 70 mm chest x-ray films were reported to be normal.

Those approached as to the significance of these findings offered different opinions, and a cursory search of the literature suggested that there was not general agreement on the matter. I was not satisfied that these were transient, or incidental findings and in May 1965 called back the men for re-examination. The rales were still present in 10 of them, and large chest films showed some degree of abnormality to be present in each case, which perhaps was not obvious on the previous 70 mm films.

Similar examination of 17 men who had been asbestos sprayers in the Dockyard revealed that three of them had asbestosis and were receiving disability pensions under the Pneumoconiosis Scheme, and that five others showed clinical and x-ray changes suggestive of asbestosis.

It was obviously desirable to investigate the problem more thoroughly, and, on discussing the matter with Dr. G. Sheers at the Plymouth Chest Clinic, I discovered that he had been finding an increasing number of cases of unexplained radiological and pulmonary changes occurring in persons attending for Mass Radiography or at the Clinic. The one thing these persons had in common was that they all worked in the Dockyard, and he wondered if the changes were due to exposure to a common harmful substance. Because of the

variety of trades followed by these men it was not immediately obvious that they had been exposed to asbestos, but this slowly revealed itself as the most likely cause.

After a chance meeting with Professor R.S.F. Schilling of the London School of Hygiene and Tropical Medicine, representations were made to the Medical Director General (Naval) who decided that research into the problems associated with the use of asbestos in Naval Dockyards should be undertaken.

Early in 1966 a research unit was set up in Devonport Dockyard with equipment provided by the Medical Research Council Pneumoconiosis Research Unit, and the results from this comprehensive study of the hazards associated with asbestos in Dockyard workers form the basis of my report.

The author has been responsible for the work described in this report. Any contributions to the study that have been made by other persons are fully acknowledged in the text.

REVIEW OF THE LITERATURE

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Asbestosis*

The heat resisting properties of asbestos have been known for over 2,000 years, but the first description of a disease process associated with the inhalation of asbestos dust was the report of a case of pulmonary fibrosis in an asbestos textile worker presented by Dr. H. Montague Murray in 1900. Murray gave evidence about this patient to the Departmental Committee on Compensation for Industrial Diseases in 1906. The patient, who was 33 years old when he died, had worked for 10 years in the carding room of an asbestos textile factory, and then another 4 years in a less dusty job. His dyspnoea was far more incapacitating than his comparatively few physical signs led Murray to expect, and at post mortem there was widespread pulmonary fibrosis with no evidence of tuberculosis. There were marked pleuritic adhesions. This patient was the sole survivor of 10 men working in the carding room when he commenced work; the others had all died about the age of thirty.

Cooke in 1924, and again with McDonald and Oliver in 1927 reported a case of asbestosis in a young tuberculos female asbestos worker and described the foreign bodies resembling asbestos fibre which were found in the lung tissue. Simson (1928) described similar bodies in the lungs of African asbestos miners and Gloyne (1929) suggested that they should be called asbestos bodies, as the core was an asbestos fibre.

*The literature relating to asbestos and malignant disease is reviewed in the section dealing with proportional mortality (page 279); that relating to dust concentrations in the section dealing with that subject (page 84).

Seiler (1928) reported the case of an asbestos worker with pulmonary fibrosis for which no other occupational or infective cause could be found. As a result of this report the Factory Department of the Home Office undertook an investigation into the effects of asbestos dust on the lungs. Merewether and Price (1930) showed that 95 of the 363 asbestos textile workers examined had definite pulmonary fibrosis due to asbestos dust. They found a correlation between the incidence of the disease and the duration and intensity of dust exposure.

Merewether examined 133 workers radiologically and 62 of them had radiological signs of diffuse fibrosis while a further 25 had films suggestive of early fibrosis. The comparatively poor standard of equipment and technique suggests that these numbers were probably an under estimate. Merewether did not find evidence of an outstanding tendency to pulmonary tuberculosis in the asbestos workers in general, or in those with asbestosis in particular.

The report suggested a dose response relationship to the development of asbestosis and that with continuous high exposure to asbestos dust the fibrosis might be fully developed in 7 to 9 years, whereas in lower dust concentrations the maturation period of fibrosis might be 15, 20 or 25 years. Merewether suggested that if this hypothesis was correct then dust suppressive measures would be vitally important in preventing the disease. The report reviewed the dust concentrations in various textile processes and recommended suitable methods of dust suppression. These measures formed the basis of the Asbestos Regulations 1931 which were not fully implemented until 1933. Asbestosis was recognised as a compensatable disease under the 1930 Workmens Compensation (Silicosis and Asbestosis) Act and a scheme was set up for periodic medical supervision of asbestos workers in certain prescribed occupations.

Burton Wood (1929) reported 15 cases of asbestosis and described the symptoms, signs and x-ray changes. The most important symptom was dyspnoea, which was progressive, and in the later stages of disease, extreme. Cough was often slight, and was a variable symptom. He noted a reduced chest expansion, cyanosis and finger clubbing often occurred. Dry crackling adventitious sounds were present, chiefly in the bases and superficially in the axillae. He thought that the latter sound might have a pleural origin. The first patient described in this series is reported as having a vital capacity of 1,600 cc and this is the first report of a lung function test in asbestosis. The x-ray films were described, and Burton Wood mentioned that although some of the cases showed a "ground glass" appearance over the lower zones, a fine mottling was seen on closer inspection. Basal pleurisy was seen in many films and also thickening of the apical pleural cap.

Merewether (1930 a & b) gave a more detailed account of the clinical findings of the Home Office survey carried out in 1928-29. He described impairment of percussion note and the reduction of chest expansion as important physical signs of asbestosis, together with scattered fine rales and clicks at the bases and axillae. Pleural crepitations and a pleural rub were heard, and one case had a pleural effusion. Well marked finger clubbing was seen in a few cases, but cyanosis was present in 5% of the persons examined. Cough, sputum and dyspnoea were the commonest symptoms and he mentioned the insidiousness of the disease.

The radiological changes were described as occurring in four stages: Stage I increased liner striations; II fairly definite fine dusty stipulation; III coarser mottling with increased linear striations; IV gross lesions with pleural changes and displacements due to the pull of fibrosing lesions.

Merewether described the essential histological lesion as a chronic, interstitial fibrosis of the lungs together with the presence of "curious" or "asbestosis" bodies. He mentioned the work of Cooke and Gloyne who demonstrated the asbestos fibre as the core of these bodies.

It is interesting to note that 10 workers gave a history of pleurisy, 8 of them since working with asbestos, and that three of the latter showed signs of pulmonary fibrosis.

The pleural changes in asbestosis were described by Gloyne (1933) who reported the common finding of old, tough, pleural adhesions. Sessile plaques were usually more marked and thicker over the lower zones. As the thickening increased it gave a "ground glass" look to the pleura, which became stiff, yellow and horn like in the thickest parts. He noted recent patches of inflammation covering and obscuring the old pleural lesions, but stated that effusions were rare because of the obliterative character of the disease.

Gloyne found "asbestosis" bodies and asbestos fibres in the lungs of persons with asbestosis, and asbestos fibres were readily found in the upper respiratory tract of asbestos workers. He noted that slight asbestosis was sometimes found in workers whose industrial history did not suggest asbestos exposure. As examples he mentioned a metallurgical research worker, and a boiler riveter.

In other countries, Marchand and Riesel (1906) had reported crystals in the lungs of asbestos workers in France, and Fahr (1914) presented micro-photographs and specimens of a case of pneumoconiosis in a German asbestos worker. Buttner-Wobst and Trillitzsch (1931) described nine cases of asbestosis in two Dresden asbestos factories and Kruger, Rostoski and Saupe (1931) reported 30 cases of asbestosis amongst 52 workers examined in Dresden. They noted that signs of old pleurisy were frequent, and that in their study moderately severe

asbestosis took 5 years to develop while all workers with more than 10 years exposure had the disease.

Merewether (1933) in a Memorandum on asbestosis reviewed the state of knowledge of the disease at that time. He again pointed out the difficulty of making the diagnosis and the insidiousness of asbestosis. He noted that attacks of dry pleurisy were common. Merewether suggested that while asbestosis was unusual in those persons exposed to the dust for less than five years, those exposed to dense concentrations for a short period would inevitably develop profound fibrosis provided that they lived long enough. A certain minimum "fibrosis producing amount" of asbestos must be retained in the lung in order to produce disabling fibrosis, and a certain "maturation" period must elapse before the development of that degree of fibrosis. He suggested that if dust concentrations could be kept below a "dust datum" level that the development of a disabling degree of asbestosis would not occur over an average working lifetime. The "dust datum" level was the amount of dust produced by flyer spinning without dust exhaust. Tables showed that the average exposure required to produce fatal fibrosis was 13.3 years and the shortest exposure was 4.4 years. The average age of death for men with asbestosis was 40.8 years while for those with silicosis it was 54.1 years. Seventeen of the 100 men with asbestosis discovered in the 1928 Home Office inquiry had died. Ten died with asbestosis, 6 from asbestosis and tuberculosis and one with asbestosis and carcinoma of the pancreas.

Wood and Gloyne (1934) reviewed 100 cases of asbestosis, and of the 12 deaths in this group two had lung cancer and one had deposits of growth in the pleura. Dyspnoea was so great as to suggest that asbestosis was a mono-symptomatic disease. They reported some other occupations which had not

previously been associated with asbestosis and gave as examples a man who had handled asbestos mattresses in the open air on an aerodrome, and a man working in a shop where asbestos was used to lag pipes.

Lanza, McConnell and Fehnel (1935) described a survey in Canada and USA in asbestos mines, mills and manufacturing plants. They examined 126 men and 67 of these had radiological signs of asbestosis, with doubtful x-ray changes in a further 37 men. It was not possible to correlate the development of the disease with definite values of dust exposure because of the frequency of job changing and the lack of retrospective dust data. The authors thought that asbestosis was clinically a milder disease than silicosis.

Shull (1936) presented a review of 71 cases of asbestosis with an average age of 34.4 years and the length of exposure ranged from 6 months to 21 years. Among the radiographic signs that Shull considered typical of asbestosis were a diffuse fine to coarse fibrosis reaching to the periphery, interstitial in character, differentiating it from the radiographic appearance of silicosis; increased density of all pleural markings; shaggy appearance of cardiac outline and a tendency towards right cardiac enlargement.

Dreessen et al (1938) published a study of American asbestos textile plants, and they suggested that if the dust concentrations could be kept below 5 mppcf new cases of asbestosis would probably not arise. They based this level on their study in which only 3 doubtful cases of asbestosis were discovered in men working in dust concentrations of less than 5 mppcf. It was unfortunate that of the 511 men examined only 66 had worked with asbestos for more than 10 years; 333 men had worked with asbestos for less than 5 years, and it is probable that the reason for the low incidence of asbestosis was in fact due to the short period of exposure. In addition to this 150 men had been

dismissed from the plants before the survey started as they were suspected cases of asbestosis and this obviously further reduced the value of the study.

Silicosis and Asbestosis (1938) edited by Lanza gives a very good survey of the accumulated knowledge of the clinical, radiological and pathological aspects of asbestosis throughout the world at that time. In the section on radiology Pendergrass drew attention to the marked thickening of the pleura in cases of asbestosis at autopsy and suggested that this thickening probably explained the haziness of the lower lung fields seen on x-ray films. He had reservations about the early radiological diagnosis of asbestosis.

Wegelius (1947) described the changes in 126 cases of asbestosis found amongst 476 workers in asbestos mines and factories in Finland. Ninety-four were classified as Stage I asbestosis - very fine network in the middle of the basal fields; 23 as Stages II and III (II - denser picture with numerous small nodules, III - marked shadowing of middle and lower fields with confused heart shadow); and 9 advanced Stage III. Pleural and pericardial thickening was present in 36% of all cases, and in all 9 with advanced Stage III asbestosis.

Luton, Champeix and Faure (1947) studied the significance of "asbestosis" bodies in the sputum of asbestos workers. These bodies were difficult to find in the early months of exposure because there was usually no sputum unless the men had a chest infection. They eventually found the bodies in all of the 162 men observed over 4 years, but none of the men had evidence of asbestosis. They showed that the bodies persisted in the sputum after exposure to asbestos dust ceased.

Wyers (1946) presented a thesis "That legislative measures have proved generally effective in the control of asbestosis". It seems that Wyers was a little too optimistic in his conclusions, but his study shows what dramatic improvements were made after the introduction of the Asbestos Industry

Regulations in 1931. He made several important observations about the processes involving the use of asbestos which were not covered by the Regulations and pointed out that persons so exposed to the dust were developing asbestosis. He observed that the function of the Medical Boards was to award compensation and not to prevent the disease.

Wyers suggested that dyspnoea was the main symptom of asbestosis, with a little cough and mucoid sputum. Chest pain occurred and was described as a dull ache. He thought that the dry rales heard at the bases were evanescent early in the disease, but persisted in the more advanced cases. He mentioned finger clubbing in 14 of the 29 cases of asbestosis and thought that it occurred early in the disease. Together with an accentuated second pulmonary heart sound and a raised diastolic pressure, Wyers suggested that clubbing made up a triad of cardiovascular signs in asbestosis. He suggested that interstitial fibrosis would interfere with gaseous exchange in the lungs causing cyanosis and dyspnoea. The disease was often uncovered and exacerbated by intercurrent chest disease. He thought that once fibrosis had started it would continue but removal from further dust exposure would slow down the rate of progress of the disease.

Wyers considered that asbestosis was a clinical rather than a radiographical disease and that the x-ray change should be used to confirm the clinical findings. The disease was more insidious and less acute than in the past and he suggested that the ground glass appearance which might have been partly due to inferior materials and technique, was seen on closer examination to be innumerable small opacities in the form of a reticulum. The lower zones were first affected and the late changes included shaggy heart and diaphragmatic outlines and blunting of the costophrenic angles. He speculated that as the disease had changed from an acute to a chronic disease, it might

emerge as a neoplastic disease as lower dust concentrations enabled people to live long enough to develop malignant tumours. In 98 cases with asbestosis Wyers described 15 cases of bronchial carcinoma, one pleural endothelioma, and 5 carcinomata of various sites.

Wyers concluded that the preventive measures had been successful up to a point, but that the disease which had already exhibited elusiveness and flexibility would become more insidious and require greater diagnostic skill.

Meikeljohn (1956) reviewed the uses of asbestos, the pathology, x-ray changes and clinical picture of asbestosis. He stated that while considerable emphasis had often been laid on the showers of persistent fine basal crepitations, he did not think that they necessarily indicated disease because they could be detected in workers within a few weeks of starting work with asbestos. He thought the most likely explanation to be that they were due to the obstruction of the bronchioles by asbestos fibre and he suggested that supporting evidence was the occurrence of altered fibres in the sputum. I cannot find anything in the literature which supports this concept.

Kivilouto (1960) reviewed the literature on pleural fibrosis and calcification associated with asbestos exposure and described his study of calcified pleural plaques. He showed that these occurred in non-occupationally exposed persons and suggested that they were caused by the penetration of the visceral pleura by asbestos fibres which then scratched the parietal pleura during the movement of the lung giving rise to mechanical irritation as the forerunner of the plaque.

Hurwitz (1961) described the radiological changes in asbestosis stating that in South Africa the pleural changes far outnumbered the cases of lower zone

parenchymal fibrosis reported by previous workers. Localized recent basal pleuritis was often seen, but not obvious pleural effusion. Extensive pleural thickening explained the poor definition of the cardiac and diaphragmatic outlines and caused irregularity and tenting of the diaphragm. He described the various forms of calcified pleural plaques in asbestos exposed workers and explained that these changes were often present alone, but they could also occur with the pulmonary fibrosis associated with asbestosis.

Shanks and Kerley (1962) suggested that the "ground glass" appearance was probably the result of fine pin point opacities, but that the first radiological sign of asbestosis was the presence of long strands of fine fibrous tissue at the bases. Occasionally extensive pleural thickening obscuring the lower lung fields was seen.

Wagner (1961) reviewed the literature and described the pathology of asbestosis. Thickened pleura was found in 103 out of 180 necropsy specimens from cases of asbestosis. Eisenstadt (1962) in the first of three papers (1964, 1965) reported benign asbestos pleurisy in an insulator. He suggested that exposure to asbestos dust in concentrations which were too low to cause asbestosis might give rise to benign asbestos pleurisy, and mentioned that it was not clear if these benign reactions were the forerunners of later mesotheliomata.

Caplan et al (1965) found radiological pleural thickening in 63% and pleural calcification in 15% of the 114 x-rays of asbestos workers. Bohlig (1965) and Sluis Cremer and Theron (1965) discussed the radiological classification of asbestosis and they both mention the need to include symbols for pleural thickening and calcification.

Smither (1965) reported the secular changes in asbestosis in an asbestos factory previously studied by Wyers. He compared series of cases from 1930-34;

1949; 1958-59 and 1960-64. The figures showed an increase in the duration of exposure needed to produce the disease from 7 years in 1930 to 17.5 years in 1960-64, and this was considered to be due to the improvement in dust control. It is interesting to note that Hunter (1955) reported basal rales "sometimes", Wyers (1949) generally, but evanescent in early cases; they were present in all the cases in the 1958-59 series, and in 96% of the 1960-64 series. The number of persons with finger clubbing also increased in the later series. Smither warned of the difficulty of interpreting a single chest x-ray, and suggested that a series of chest x-rays was of much greater value in the early diagnosis of asbestosis. He drew attention to the increasing number of cases with malignant disease.

Krige (1966) described the radiological changes in asbestosis in South Africa reporting that bilateral pleural thickening of the lateral chest wall was a common manifestation. The thickening could resemble an encysted effusion, but calcification was a late manifestation. Hourihane et al (1966) presented a study of 100 cases of hyaline and calcified pleural plaques in persons exposed to asbestos and reported that 85% of plaques seen at necropsy were invisible on posterior anterior chest radiographs. The 15% of plaques visible on x-ray films were extensive calcified lesions associated with histological interstitial fibrosis. In contrast, 29 of the 31 plaques found only at necropsy did not have interstitial fibrosis. The authors suggested that radiologically invisible plaques were probably the result of casual small, environmental exposure to asbestos, while the plaques detectable by radiology were large, often accompanied by asbestosis and were the result of industrial exposure. In another paper Hourihane and McCaughey (1966) discussed the pathology of asbestosis and described it as being essentially a peribronchiolar

fibrosis obliterating the surrounding alveoli as it extends out from the bronchioles. The pleura in cases of asbestosis was nearly always abnormal. Hyaline plaques were the most common pleural manifestation. Rusby (1968) described the pleural changes associated with asbestos exposure in 5 cases with calcified pleural plaques. He suggested that pleural calcification might predispose to the development of mesothelioma of the pleura or peritoneum.

Meurman (1966) reported bilateral pleural plaques in 39% of routine autopsies in Finland and suggested that most of these resulted from casual environmental exposure to asbestos.

The Factory Inspectorate Memorandum on the Problems arising from the use of Asbestos (1967), described the types of asbestos in use and the main uses for asbestos materials in this country, together with a brief description of the population thought to be exposed to the dust. The increasing number of cases of asbestosis was noted, especially in men not protected by the regulations. Suggestions were made to extend the scope of the regulations, and the medical supervision of workers. The Ministry of Social Security (1967) published a booklet on Pneumoconiosis and allied occupational chest diseases in which the criteria for the diagnosis of asbestosis were described. Given a history of exposure to asbestos the publication suggested that the presence of any two of the following factors would be strongly suggestive of asbestosis. The factors were dyspnoea; clubbing of the fingers; basal rales and crepitations; radiological changes; and reduced transfer factor (carbon monoxide). It was also suggested in this booklet that there are two types of disease; one affecting the lung parenchyma, and the other, the pleura.

The Digest of Pneumoconiosis Statistics (1967) showed that the numbers of cases of asbestosis diagnosed by the Medical Boards increased from 52 in

1962; to 83 in 1964; 114 in 1966 and 168 in 1967. In addition there were 112 examinations in 1967 where the disease was not diagnosed in claimants appearing before the Boards.

Collins (1967) described the findings in 22 men exposed to asbestos. Ten of these had pleural changes, and in two of them pleural effusions occurred, first on one side of the chest, and then the other. The second pleural effusion occurred in one case while he was having antituberculous treatment, and in the other case it developed after a course of antituberculous treatment. Collins suggested that these were possibly cases of benign asbestos pleurisies. The same author (1968) reported a high incidence of pleural reactions in asbestos workers in Durban. He suggested that these lesions might be the precursors of calcified pleural plaques and might be seen more frequently in persons exposed to relatively low asbestos dust concentrations. Eleven cases of pleural reaction were found in 522 asbestos workers (2%) in comparison with 0.6% in workers not exposed to asbestos.

The effects of asbestos exposure on insulation workers

Probably the first report of the effects of asbestos on laggers in the shipbuilding industry was that of Alden and Howell (1944) when they reported that 99 men out of 167 examined had asbestos corns on their hands. They did not confirm asbestosis in any of these men.

In 1945 the Chief Inspector of Factories of the Ministry of Labour and National Service issued a warning to the shipbuilding and shiprepairing industry about the danger to health from the increasing use of asbestos in ships*. The letter explained that while asbestos dust had no immediate serious effects, serious results were apt to develop later. Among other suggestions, he advised better ventilation, damping the dust, and cleaning up the asbestos

*See page 322.

debris quickly. He also advised the use of approved dust respirators for men fitting or removing any dry asbestos insulating material and during the spraying of asbestos.

Fleischer et al (1946) carried out a survey of four US Naval Shipyards and despite high dust concentrations they found only 3 cases of asbestosis out of 1,074 men examined. They concluded that pipe covering in the shipbuilding industry was not hazardous, but only 54 men in this survey had been exposed for more than 10 years and the only 3 men who had been exposed for more than 20 years were the men who had the disease.

Frost et al (1956) examined 31 asbestos insulators who had been exposed to the dust for more than 20 years. There were x-ray abnormalities in 22 of these men with definite asbestosis in 9 cases. Pleural abnormalities were found in 19 cases, ranging from obliterated costophrenic angles and pleural adhesions to extensive pleural thickening and calcification. In a few cases the pleural changes were so widespread that no diagnosis could be made of the condition of the underlying lung. The authors suggested that pleural calcification might be a new feature of asbestosis, as most of the earlier literature had referred to fibrous pleural thickening. They mention the work of Smith (1952) in which she found pleural calcification more commonly than expected in talc workers, but not in any of the 261 asbestos workers examined. Frost pointed out that all the men in their study had also been exposed to dusts other than asbestos, Kieselguhr, magnesium, rock wool and glass wool, and that perhaps the changes might be due to mixed dust exposure.

Walters (1959) in his account of the uses of asbestos in a Naval Dockyard reported that no cases of asbestosis had been discovered among asbestos sprayers for the 12 years up to 1958, but noted that one of them had a pleural effusion which had resolved. He described the difficulty of making the

diagnosis of asbestosis and reported one case of asbestosis among pipe laggers, and six cases in which the diagnosis was suspected radiologically but not confirmed by the Pneumoconiosis Medical Panel. Leathart (1960) presented a detailed study of 10 cases of asbestosis in insulators and compared the findings with those from a group of 11 pipe laggers who did not have the disease. The diagnosis of asbestosis was made on a history of asbestos exposure and radiological evidence of the disease. Leathart commented on the disagreement among previous authors about the significance of basal rales in asbestosis. He found basal rales in 9 of the 10 cases of asbestosis, and none of the control group, and suggested that these sounds were associated with bronchiectasis which was present in 6 of the affected men. Finger clubbing was present in 5 of the 6 men with asbestosis and bronchiectasis, and in one of the 4 men with asbestosis alone.

Leathart (1962) and Leathart and Sanderson (1963) drew attention to the fact that more cases of asbestosis were occurring despite the improvements in working conditions brought about by the Asbestos Regulations 1931. They explained that while this was in part due to the greatly increased use of asbestos, the main reason was probably because many men were exposed to the dust in the insulating trade and these men were not protected by the regulations. They suggested the use of substitute materials and an extension of the Asbestos Regulations, and the Silicosis and Asbestosis (Medical Arrangements) Scheme to cover the insulating trade. In his thesis, Leathart (1962) described 14 cases of asbestosis. Eight of these worked in Shipyards as boiler coverers. All the patients with asbestosis had basal rales, and they were all disabled to a varying degree by dyspnoea.

Marr (1964) described the asbestos materials and insulating processes in

Long Beach Naval Shipyard USA. Five men out of 60-80 insulators working mainly in the ships were compensated for asbestosis and one had died of the disease. He suggested that Fleischer's (1946) report was misleading as in that study few men had been exposed to the dust for 20 years. Marr stated that many of the men in his study had been exposed to asbestos for more than 20 years, but he does not give sufficient data in this report to give more than an impression that a hazard existed.

A comprehensive list of occupations involving exposure to asbestos was given by Hueper (1965). Selikoff et al (1965) drew attention to the widespread asbestos exposure of insulation workers and described an investigation of 1,117 persons almost half of whom had asbestosis. The percentage of abnormal x-ray films rose from 10% of those with less than 10 years from first exposure, to 73% of those with 20-29 years from first exposure and 94% with more than 40 years from first exposure to the dust. In another paper Selikoff (1965) described the occurrence of pleural calcification in 150 of the 1,117 men examined. The percentage affected rose with increased time since first exposure and rarely before 20 years since first exposure. Pleural fibrosis was noted in 192 men and appeared after shorter exposure than pleural calcification. Of 802 men with less than 30 years from first exposure, 13 had calcified pleural plaques and 65 had pleural fibrosis. He found unilateral pleural calcification in half the cases, and suggested that when earlier traumatic or infective causes could be ruled out then the calcification was probably due to asbestos.

McVittie (1965) showed that cases of asbestosis diagnosed by the Pneumoconiosis Medical Panels were increasing in number, and that 41% of the 247 new cases diagnosed between 1955 and 1963 were employed in the insulating trade. The average duration of exposure before diagnosis for laggers was

26 years; sprayers 8.3 years and mattressmakers 11.3 years. Comparative figures for the asbestos manufacturing processes were 14 years for opening and disintegrating; 15 years for weaving and 16 years for carding, spinning and for slab and pipe making. He suggested that the diagnosis of asbestosis should be made on an adequate history of exposure to asbestos and the presence of two of the following factors; basal rales, finger clubbing, radiological appearances and lung function changes. Adequate exposure was, in some occupations, three or four years of recent exposure, but generally some eight years exposure were required. In some cases very short past exposures 20 years previously might be adequate. McVittie reported that the Pneumoconiosis Medical Panels considered bilateral pleural plaques as almost diagnostic of asbestosis.

Elmes (1966) also drew attention to the asbestos exposure of insulators and discussed the symptoms and signs of the disease. Thirteen of a group of 21 insulating workers with early asbestosis had dry basal rales, 16 had asbestos corns on the hands, 7 clubbing of the fingers and only 4 had dyspnoea of grade 2 (MRC standard) or more. He described two forms of pleural thickening associated with asbestosis. A diffuse lower zone thickening with ill defined margins and related in severity to the parenchymal change, and circumscribed areas of hyaline fibrous tissue on the diaphragm, pericardium or chest wall.

Anton (1967) reported 12 cases of multiple pleural plaques in men exposed to asbestos in shipyards, and in 1968 described a further 28 cases. Non-calcified plaques were usually seen at the periphery of the lung fields and he thought they played an important part in the diagnosis of asbestos exposure. Conditions had improved in asbestos factories and Anton suggested that because of this a milder form of asbestosis might be prevalent, and the presence of the

pleural plaques might be one sign of this milder disease. He considered that pleural fibrosis might have been responsible for the earlier workers description of ground glass appearance in the lower lung fields. Leathart (1968a) referred to Anton's (1967) paper and noted that 4 of the 12 cases were described as having plaques of soft tissue density. Leathart then reported 60 such plaques in 127 persons exposed to asbestos (71 with certified asbestosis) and stated that in his experience the hyaline plaques were much more common than the calcified variety. The plaques were best seen at the periphery of the lung fields and oblique screening showed that they lay postero-laterally on the chest walls. There was post mortem confirmation of the plaques in two cases. Radiological evidence of pulmonary fibrosis was present in only 36 of the 60 cases with pleural plaques and Leathart suggested that the plaques might sometimes be the only radiographic evidence of pulmonary asbestosis for which there might be clinical, physiological or biopsy evidence. However, in a later report Leathart (1968b) concluded that hyaline pleural thickening occurring without radiological evidence of pulmonary fibrosis had no specific effect on lung function.

Sheers and Templeton (1968) carried out a radiographic survey of every tenth person employed in a Naval Dockyard. Of 1,414 persons examined who had varying exposure to asbestos, 4 were found to have pulmonary fibrosis due to asbestosis, 11 had extensive pleural fibrosis and 48 presented with limited pleural plaques. The prevalence of abnormalities increased with the length of time since first exposure and also with the degree of exposure in various occupational groups. The authors suggested that the pleural changes predominated over the parenchymal disease because the intermittent exposure to asbestos was insufficient to cause asbestosis except in the high risk groups.

Harries et al (1969) have carried out an extension of this survey in three other Naval Dockyards with essentially similar results.

Lung function in asbestosis

Leathart (1962) presented a comprehensive review of the literature on the effects of asbestosis on lung function up until 1957. He summarized the results of published tests of lung function in cases of asbestosis and showed that a reduction in vital capacity and diffusing capacity for carbon monoxide or oxygen, desaturation of arterial blood and an increase in the ventilation equivalent for exercise were the defects commonly associated with the disease. The changes appeared to relate well to clinical abnormality, but not to the radiological appearances.

Previously Leathart (1960) had presented a detailed study of 10 cases of asbestosis and compared them with 11 laggers without radiological evidence of the disease. He demonstrated significant reduction in diffusing capacity for carbon monoxide (steady state method), vital capacity, compliance and maximum voluntary ventilation in the group with asbestosis. There was no evidence in this study that lung function defects preceded the radiological changes, but the diffusing capacity of the control group was at the lower limit of normal.

Williams and Hugh Jones (1960a) examined 40 patients, 21 of whom were certified as having asbestosis by the Pneumoconiosis Panel. Ten of the others had worked in the same asbestos factory as the certified cases but showed no radiological signs of the disease, and the remaining 9 were cases where the diagnosis of asbestosis was in doubt. The x-ray changes were scored according to the degree of mottling present, although in another paper (1960b) the authors discussed the difficulty of repeatable reading of x-ray films in asbestosis. Basal crepitations were found in all the 21 men with asbestosis, dyspnoea was present in 20 and finger clubbing in 17 of them.

There was a reduction of inspiratory capacity, hyperventilation on exercise with no evidence of airway obstruction and a lowered diffusion capacity (single breath carbon monoxide) in the patients with asbestosis. In this group there was good correlation between diffusing capacity and the severity of dyspnoea and also between finger clubbing and the grade of dyspnoea. There was a significant correlation between the reduction in diffusing capacity and degree of radiological change. Four of the 10 radiologically normal asbestos workers had reduced diffusing capacity and three of these had basal crepitations. The third group of 9 cases of questionable asbestosis showed results which strongly suggested asbestosis in 6 of them. All of these six cases had a reduced diffusing capacity with a well maintained ventilatory capacity and five of them had dyspnoea, clubbing and basal crepitations as well as doubtfully abnormal x-ray films. The authors suggested that the diffusion defect was a constant finding in established cases of asbestosis, that it correlated well with the grade of dyspnoea and x-ray changes, and that the changes in diffusing capacity might precede clinical or radiological signs of the disease. It is interesting to note that there were abnormal electrophonetic patterns in the sera of 13 of the 21 men with asbestosis.

Heard and Williams (1961) described the post mortem histological findings in six men with asbestosis and correlated these with the previously assessed lung function changes. All six men had pleural adhesions and three had thick pleural plaques. Pulmonary fibrosis was present in all, but only to a slight degree in one case who had a carcinoma of the bronchus. Histological emphysema was present in one case who had functional changes suggesting this condition during life. Very slight emphysema was reported in two other cases. The lung function tests in all of them except the patient with clinical emphysema showed a pure diffusing defect with a reduced carbon monoxide

diffusing capacity, hyperventilation and desaturation on exercise and no evidence of airways obstruction. The authors speculate on whether or not the restrictive lung changes with reduced inspiratory capacity and decreased lung volume were due to pleural adhesions and plaques, or whether the associated pulmonary fibrosis was the most important cause.

Bader et al (1961) examined 17 men with asbestosis and showed some reduction in vital capacity in half of them. In 4 of 5 men tested there was a reduction in the diffusing capacity for oxygen, but this and other tests of lung function correlated poorly with the degree and duration of exposure to asbestos.

Leathart (1962) presented a very detailed study of lung function findings in 12 male cases of asbestosis and 11 radiologically normal male insulators. This was an extension of the work in which he had previously discussed the clinical, radiological and lung function features of the disease. (Leathart 1960). He found significantly abnormal results in 9 of the 10 tests of lung function in the cases of asbestosis. The useful tests were, maximum voluntary ventilation, dead-space effect, diffusing capacity, functional uptake of carbon monoxide, dynamic compliance, non-elastic work of breathing, vital capacity, and expired concentration of CO_2 during maximum voluntary ventilation. The last test was devised by Leathart and he suggested that the results indicated the presence of defective intra-pulmonary mixing and gave a rough idea of its degree. He suggested that in asbestosis uneven alveolar perfusion existed. This might have contributed, in part, to the reduced diffusing capacity measured by the steady state method.

A marked reduction in dynamic compliance was found in the cases of

asbestosis, and as this correlated well to the reduction in vital capacity Leathart suggested that the latter test would be useful to perform serially on workpeople exposed to asbestos. In an interesting discussion on the reasons for the reduction in ventilatory capacity found in patients with asbestosis Leathart thought that the relevant defect might possibly lie in the pleura, or the joints and muscles that move during respiration. He devised a test designed to measure the mean intra-thoracic pressure or "resistance index", which was the average pressure (in respect to volume) during the first 0.75 seconds of a forced expiration. He thought that the simultaneous presence of a low indirect MBC with a low "resistance index" indicated a defect in the parieties because it ruled out the reduction in MBC due to increased air flow resistance. Increased parietal friction due to pleural fibrosis might occur in asbestosis, and though this was suggested in one case in which these defects were found there was no evidence of pleural change in another with similar findings.

Leathart suggested that a restrictive ventilatory defect probably caused the dyspnoea in those cases who also had bronchitis, and that reduced diffusing capacity limited exertion in those with "pure" asbestosis. He discussed the use of pulmonary function tests as an aid to the diagnosis of asbestosis and after pointing out that the tests were not in themselves diagnostic, suggested that low pulmonary compliance was the outstanding feature of asbestosis. His study did not show that fibrosis of the lungs could be detected by lung function tests before changes appeared on chest radiographs.

Bjure et al (1964) reported lung function studies on 8 asbestos workers with radiographic asbestosis, and 6 radiographically normal men exposed to glass wool. Pleural thickening was present in all of the 8 cases of asbestosis and pulmonary streaking or mottling in 6 of them. Highly significant reductions

in vital capacity, $FEV_{1.0}$, and steady state CO diffusing capacity were reported in the cases of asbestosis. The men exposed to glass wool had normal values. The authors suggested that the diffusion defect was partly due to impaired diffusion and partly due to disturbance of the ventilation/perfusion ratio. Six of the eight asbestos workers showed increased pulmonary vascular resistance, which, in the absence of obstructive lung disease, suggested a reduced pulmonary vascular bed due to fibrosis.

Thomson et al (1965) examined 28 asbestos workers, 19 of whom had been certified as having asbestosis. The authors suggested that x-ray changes, basal rales and dyspnoea were outstanding in their ability to differentiate between certified and uncertified cases of asbestosis. In a discriminant analysis of the lung function tests carried out on these subjects the authors showed that the inspiratory capacity, vital capacity, diffusing capacity for carbon monoxide (single breath method), and compliance were the best discriminators. There was no evidence of airways obstruction in these patients.

Hunt (1965) described a study of 450 workers exposed to asbestos and 260 control subjects from the same factory. He demonstrated a reduction in gas transfer (diffusing capacity for carbon monoxide estimated by the single breath method) accompanied by a fall in vital capacity in 110 workers, and a fall to below 60% of predicted normal value of gas transfer in a further 36 workers exposed to asbestos. He suggested that lung function studies could detect asbestosis before the changes were apparent on chest radiographs.

Bader et al (1965) reported serial lung function studies of 13 asbestos workers over a 10 year period. The authors found a fall in vital capacity in 12 of them and thought this correlated well with the progression of x-ray changes and degree of breathlessness.

Leathart (1965) presented the results of measurements of compliance,

diffusing capacity and vital capacity in three groups of asbestos workers. Thirty were insulation workers without any sign of asbestosis; 41 were cases of asbestosis; and 6 men had pleural calcification. The mean values for these tests were lowest in the group with asbestosis and highest in those with pleural calcification. No test discriminated absolutely between the group with asbestosis and those without, but compliance seemed to be the best discriminator. Leathart suggested that changes in compliance and vital capacity should precede the reduction in diffusing capacity, but pointed out that other workers had found the reverse trend to occur. This might be due to differences between the steady state and single breath methods of estimating the diffusing capacity and Leathart suggested that an early defect detected by the latter method might be due to inequality of ventilation and perfusion as the result of patchy fibrosis rather than alveolar infiltration.

Kleinfeld et al (1966) examined 56 asbestos insulators who had more than 14 years exposure to dust, and 50 men of various unspecified occupations who were stated to have no previous exposure to asbestos. They found significant differences between the two groups for the presence of cough, crepitations and finger clubbing, and also for the mean values of vital capacity, total lung capacity and the single breath carbon monoxide diffusing capacity. None of the control group had x-ray abnormalities, but 21 of the asbestos workers showed some degree of pulmonary mottling, 16 had obliteration of the costophrenic angles and 10 showed some obliteration of the cardiac border. Five insulators had pleural calcification.

A comparison of the 16 men with pulmonary nodulation and 20 men with normal x-rays showed no significant differences of age, years of exposure or for symptoms or signs. There were significant reductions in vital capacity,

total lung capacity and diffusing capacity in the men with radiological mottling. The authors concluded that while an appreciable number of asbestos workers had significantly reduced lung function tests there was poor correlation between lung function and clinical and radiological findings of the exposed group as a whole, and there was no correlation between the duration of exposure and lung function findings in this group. They did not report the exposure history of the 17 asbestos workers whose vital capacity fell below the 95% confidence limit, nor for the 13 men whose diffusion capacity fell below this limit. This would have been an interesting comparison within the group especially as none of the values for those tests in the control group fell below these levels.

Gandevia (1966) suggested that serial lung function tests were likely to be more useful than serial chest radiographs in the supervision of asbestos workers. In a comparative study of 41 male asbestos workers and a control group, he showed that the asbestos workers had a greater decrease in $FEV_{1.0}$ and possibly in vital capacity with an increase in standardized ventilation over a period of three years than the control subjects.

Leathart (1968c) described the lung function results in 181 asbestos workers and concluded that asbestosis was usually, but not always, associated with a loss of compliance and diffusing capacity while pleural calcification alone had no effect on lung function. He presented serial measurements of compliance, vital capacity and diffusing capacity in 14 cases of asbestosis over periods of 2 to 9 years. The mean reduction over the period for diffusing capacity was 26%; vital capacity 16% and compliance 19%. Three patients who also had rheumatoid arthritis deteriorated more quickly than the others. Serial measurements of steady state carbon monoxide diffusing capacity were

presented for 12 laggers with normal radiographs and 5 of these showed a drop to less than 80% of the original reading. The variability of the results using the steady state method was seen in this study. Leathart suggested that the presence of basal crepitations was intermittent early in asbestosis, but a permanent feature of the developed disease, and that their detection sometimes preceded the drop in diffusing capacity in serial observations of asbestos workers. He concluded that diffusing capacity was probably the best method for making the early diagnosis of asbestosis, and that the use of vital capacity and compliance did not perhaps seem to be as useful as had previously been thought.

Becklake et al (1968) presented an interim report on a large survey of Canadian asbestos workers and showed that there was a reduction in lung volume, expiratory flow rate and diffusion capacity in men with definite early radiological evidence of asbestosis. There was close correlation between the profusion of small irregular opacities and the lung function impairment suggesting that the use of lung function tests might, after all, be of some value in the early diagnosis of the disease. There was also evidence in this survey that while pleural calcification alone appeared to have no adverse effect on lung function, diffuse pleural thickening was accompanied by a reduction in vital capacity, expiratory flow rate and diffusing capacity on exercise.

Summary and discussion

The early reports on asbestosis were made by physicians who relied on careful clinical examination of the patient, and pathologists who studied the post mortem histology. Radiology played an increasingly important part in the description of the disease as the technical qualities of the equipment and films improved.

From those early papers we see that asbestosis was a rapidly progressive

interstitial fibrosis of the lung which usually developed within 7 years exposure to the very dusty conditions in the industry at that time, and which was often accompanied by extensive pleural thickening. Pulmonary tuberculosis was commonly found in the population in general, and often occurred in persons with asbestosis. Unlike silicosis, there did not appear to be an increased incidence of tuberculosis in patients with asbestosis. Death commonly occurred within 10-14 years of starting work with asbestos and was usually as the result of cor pulmonale.

Big improvements in the working conditions of asbestos factories followed the 1930 Report by Merewether and Price. This resulted in a longer "maturation" period for the disease as we can see from the results of Myers and Smither who showed that the average period of exposure before 1931 was 7 years, in 1946 was 10.4 years and in 1965 it was 13.1 years.

Apart from a few isolated reports these papers relate to asbestosis developing in workers employed in asbestos manufacturing processes, and the Regulations applied only to this part of the industry. Insulation workers, makers of battery boxes, brake linings and many other occupations involving the use of asbestos were not protected by the Regulations. Because the exposure was intermittent and of extremely variable concentration and composition the development of the disease in most of these industries was long and insidious. Fleischer et al (1946) for instance deduced that there was not much hazard in the shipyard insulation industry, but they did not examine enough persons with long enough exposure to the dust. Frost (1956) showed that many men with more than 20 years exposure as insulators showed signs of the disease, and this has since been the experience of many workers.

As the "maturation" period of asbestosis has increased other manifestations

of the disease have appeared. The question of malignant disease is discussed in a later section of this report, but is the most sinister complication. Important changes involve the pleura. In this review I have tried to draw attention to the fact that this is not something that has only recently been noticed, but that from the very earliest reports it is clear that the pleura was involved in the disease process. It would appear that with reduced dust concentrations in the factories, and intermittent exposures elsewhere, the florid parenchymal fibrosis is less common, and less acute than it used to be, and has been replaced by an insidious, very slowly progressive fibrosis, while the changes are still occurring in the pleura. These pleural changes are more obvious because of the lesser parenchymal changes. There may also be cases where pleural fibrosis is present without any parenchymal change, but it is likely that some degree of histological pulmonary fibrosis may be present if there has been sufficient exposure to result in the formation of hyaline pleural changes.

Many of the early writers report "dry pleurisies" as being common in asbestos workers, and it is possible that we are now able to observe these pleural reactions more easily as men are more frequently examined radiographically. There is a disturbing uncertainty in the more recent reports about the significance of these pleural changes. These reactions seem to fall into three main groups, extensive pleural fibrosis with perhaps effusion which may spontaneously resolve; limited hyaline pleural plaques; and calcified pleural plaques. The questions still unanswered are whether or not these pleural changes are a part of the progressive, pulmonary fibrotic disease, or if they are changes merely confined to the pleura, or are they the forerunners of malignant disease?

The definition of the term asbestosis is of interest in this review. It

can be seen that there has been some disagreement over the presence and significance of the various symptoms, signs, radiological and lung function changes, and to the importance to be attached to the pleural changes.

The concensus of opinion which is now probably acceptable by most workers is that asbestosis is a progressive pulmonary fibrosis, as the result of exposure to asbestos dust, accompanied by fibrotic changes in the pleura which may develop areas of calcification. The disease may be complicated by bronchial carcinoma, and exposure to asbestos is also associated with the development of mesothelial tumours of the pleura or peritoneum.

The main symptoms are dyspnoea on exertion, cough with scanty mucoid sputum, and in some cases intermittent, dull chest pain. It is now accepted that dry basal rales or crepitations heard most commonly at the bases and in the axillae are characteristic of the disease, and that these sounds are constantly present when the disease is reasonably well advanced. It has been suggested that the presence of these sounds may precede the x-ray or lung function changes in some cases. Clubbing of the fingers often occurs and cyanosis is present in the advanced cases.

The lung function changes are those occurring in any interstitial fibrosis. There is a restrictive ventilatory defect with no airways obstruction (excepting late in the disease in some cases who develop bronchitis); a reduced compliance; hyper-ventilation on exercise with desaturation of arterial oxygen; and a reduction in the transfer factor.

The radiological changes include progressive irregular nodulation which is more marked over the lower lung fields, with pleural fibrosis obscuring the cardiac and diaphragmatic outlines. Hyaline and calcified plaques are often seen, without necessarily any obvious radiological parenchymal fibrosis.

"Asbestos pleurisies" are being reported more frequently and evidence is being accumulated which suggests that this is a real clinical entity.

Little advance has been made in the matter of early diagnosis of the disease which still requires intelligent assessment of all the relevant data. It is possible that the immunological studies which are now proceeding may help in the field of early diagnosis.

Diagnosis of asbestosis is also complicated by the problem of compensation for a disease related to occupation. In Britain the term Pneumoconiosis is used in the National Insurance (Industrial Injuries) Act to include the occupational dust diseases, coal miners pneumoconiosis, silicosis, and asbestosis. The definition of the disease under this Act is "fibrosis of the lungs due to silica dust, asbestos dust or any other dust and includes the condition known as dust reticulation". It is clear that under this definition the term asbestosis is restricted to pulmonary fibrosis and does not include changes occurring in the pleura without obvious parenchymal fibrosis. Mesothelioma of the pleura or peritoneum is a new prescribed disease (No. 44) under the Act, and is recognised as being associated with asbestos exposure, but not necessarily to the presence of pulmonary fibrosis.

It seems that many reports, especially the more recent ones, have not made clear their definition of the term "asbestosis". In some cases it would appear that any form of pleural fibrosis or calcification which could not be accounted for by traumatic or infective causes may have been included in the term "asbestosis".

This discussion may only be of academic interest, but I believe that with the discovery of more cases with extensive pleural reactions probably due to asbestos and which are not mesothelial tumours, the question of the legal

definition will have to be reviewed. This will be important for two reasons. Firstly, a decision might have to be taken to remove such patients from further exposure to asbestos dust resulting in loss of pay, and secondly, some of these reactions may cause actual disability, again enforcing a change of employment. In both cases the sufferer should be entitled to the same financial compensation as the person with "asbestosis".

Further discussion of the changing pattern of the disease and the problem of diagnosis will follow the presentation of the clinical studies.

There have been few reports relating to shipyard asbestos exposure (Fleischer et al; Marr; Leathart and Sanderson; Selikoff et al; Murphy and Ferris; Sheers and Templeton) and of these only the paper by Sheers and Templeton (1968) gives any idea of the incidence of asbestos diseases in the total population of a shipyard. This work, which has been confirmed by a larger study by Harries et al (1969), shows that not only the men actually using the asbestos materials have been at risk. As might be expected, many other men have been exposed to the dust, and are now developing diseases due to asbestos. The results of a detailed study of shipyard workers forms the body of this report.

A DESCRIPTION OF THE USES AND USERS OF ASBESTOS IN DEVONPORT DOCKYARD

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THE USES AND USERS OF ASBESTOS IN DEVONPORT DOCKYARD

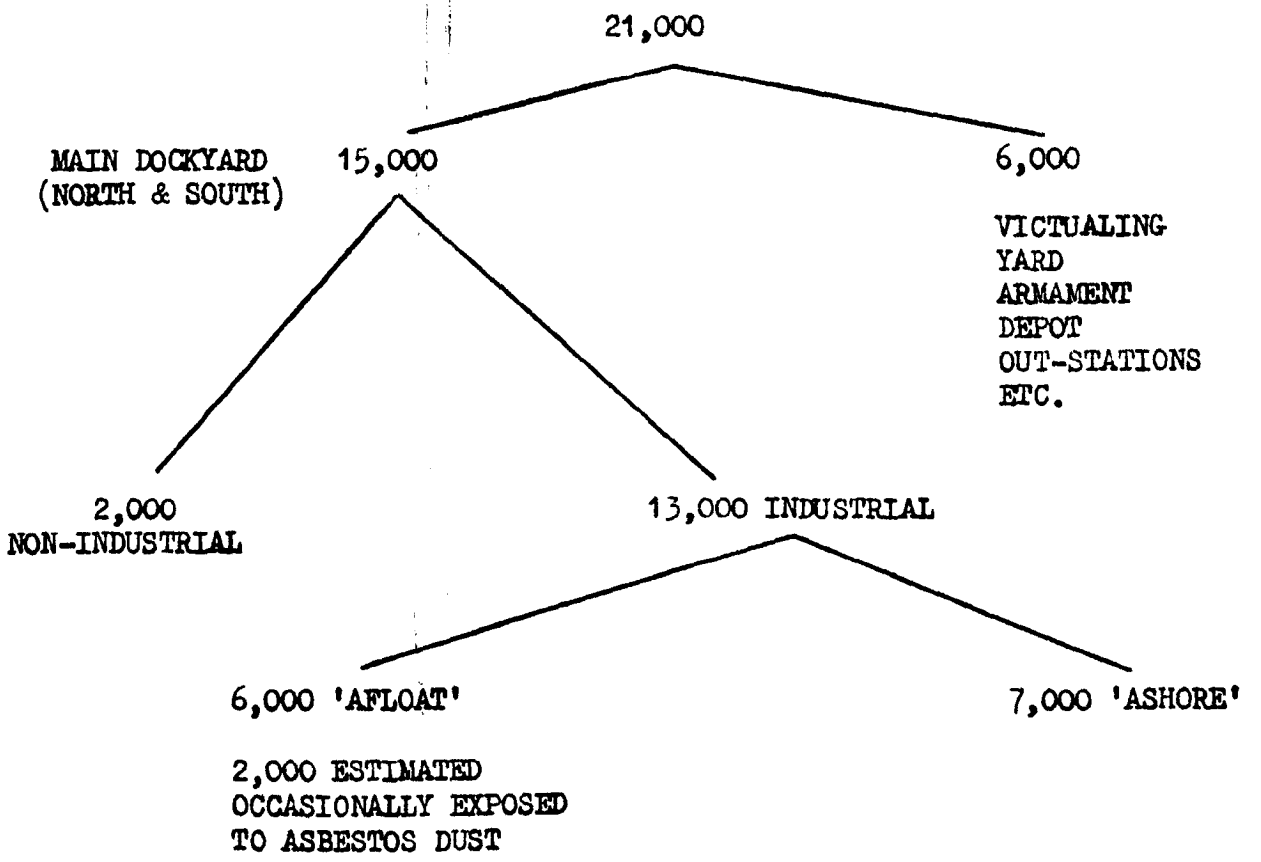
Devonport Dockyard has been building and refitting Naval Warships for over two centuries and presently employs a civilian labour force of some 21,000 employees. The main work of refitting and shipbuilding takes place in the North and South Yards in which about 15,000 men are employed. Some 6,000 of these men may be working on the ships at any one time, while the remainder work in the various workshops, factories, the building slip, and on the docksides. An official estimate of the number of men who may be continuously or intermittently exposed to asbestos dust at work is 2,000, but this may be an underestimate as any of the 6,000 men at work on the ships might have come into contact with it from time to time. (Table 2.1).

The work consists mainly of refitting and repairing ships, and while capital ships have been built in Devonport, frigates of about 2,000 tons are now usually built at the rate of one every two years. It is important to realise that not only do warships contain more machinery in smaller compartments than merchant ships, but there is a naval requirement for better insulation and thus more asbestos insulating material is used. Large ships such as Aircraft Carriers or Cruisers undergo "short refits" of up to 6 months every three years when machinery modification and repair may require the renewal of some of the insulating materials. Every 10 years, or so, a "long refit" is undertaken in which extensive modifications, modernization and repairs are carried out lasting up to three years and involving both the machinery and structure of the ships. This may entail the removal of nearly all the machinery insulation, and a large part of the

TABLE 2.I

THE ROYAL DOCKYARD, DEVONPORT

TOTAL WORKING POPULATION



environmental or structural insulation. Extensive refits of this kind are hardly ever contemplated in merchant ships, and so the civilian shipyards may have been spared the widespread high concentration of asbestos dust caused by these procedures. The smaller ships undergo a similar cycle of refits, but the refit times are obviously shorter.

Warships under sail and with wooden walls required no insulation. Wood was a poor conductor of heat and gave adequate protection from the heat of the sun. The maintenance of a reasonable environmental temperature required only simple ventilatory methods such as wind scoops and open gun ports. The advent of steam propulsion and steel hulls created problems of heat insulation of both the machinery and the steel decks.

In the 1880's machinery was insulated with preformed sections of 85% magnesia with 15% asbestos as a binder, asbestos millboard, and asbestos filled mattresses. These materials were extensively used, with large amounts of asbestos cloth, to insulate all the hot surfaces in engine rooms and boiler rooms, and were adequate at the operating temperatures below 750°F which existed up until 1950. Some of the fibre used in these materials was crocidolite because this type of fibre was required by the Admiralty specification, but cloth was always made of chrysotile.

After 1950 steam temperatures rose to 850°F and improved insulation was required. Very large pipe sections made almost wholly of amosite fibre were used, but experience showed that this was not as efficient as had been expected. There was oxidisation of the sections at the hot face, and they then tended to disintegrate with the vibration of the machinery. The joints between the sections allowed too much "wild heat" to escape into the compartments and in the early 1960's amosite sections were removed from the specifications.

At the same time hot face temperatures had risen to 950°F and it was decided to use sections of calcium silicate with 12% amosite asbestos as a binder, for pipe lagging. This is applied in two layers to avoid heat loss at the joints, and as it is brittle it is covered with asbestos cloth which in turn is coated with a hard setting asbestos cement before being painted. Asbestos filled mattresses are still used in places where the insulation has to be removed from time to time for the inspection or maintenance of machinery.

The insulation of the steel decks and hull against heat and cold is essential to maintain a reasonable environmental temperature, and, because it could also prevent the spread of fire, extensive use was made of asbestos for this purpose. From the middle of the Second World War millions of square feet of asbestos fibre was sprayed on deckheads and bulkheads of warships, and this fibre was mainly crocidolite. In the late 1950's it was found that this material was contributing a lot to the top weight of the ships and it was increasingly replaced by lighter materials, usually glass fibre. For machinery spaces cement faced asbestos board was used for deckhead insulation, and this often contained crocidolite.

All the hot and cold water pipes, ventilation trunking and chilled water lines throughout the ship were insulated by cork or felt and covered with asbestos cloth, and asbestos fibre board was used for insulating galleys, cold rooms, and to protect electrical wiring running up masts.

Many compartments in a ship are required to be insulated against noise and asbestos fibre board, acoustic tiles and perforated asbestos board has been used for this purpose. Asbestos board is also used in boiler casings. Different types of asbestos millboard are used for insulating ships' galley equipment.

Asbestos fibre has been used for gland packing, and resin bonded asbestos slabs are used in propeller shafts and periscope housings.

Cloth woven from chrysotile asbestos is used to protect electrical cables and other equipment from hot metal splashes and slag falling from welding, flame cutting, and burning processes. The cloth is also used to lay on top of electrical elements which heat steel plates up to 150°C for certain welding techniques. Welding electrodes wound with blue asbestos cord were used before and during the war.

There are other varied uses for asbestos compounds in ships, but they usually do not give rise to asbestos dust under normal working conditions.

Machinery and pipe insulation is carried out by laggers and consists of applying the pre-formed insulating sections to pipes, securing them by wire, and covering the sections with asbestos cloth which is then stitched with asbestos twine. Small pipes with difficult bends are insulated with asbestos rope. Large turbines, or other equipment with awkward shapes are covered with flat sections cut to shape, and with magnesia, or calcium silicate compound containing amosite asbestos both of which are moulded on wet after the dry material has been mixed with water. Dust is produced while cutting, breaking, or moulding into position the sections, when tearing the cloth, when applying asbestos rope, and when mixing asbestos cement.

The laggers are also responsible for removing or stripping the old insulation before the refit commences, and this process consists of cutting and tearing the overlying asbestos cloth, and ripping down the underlying sections. This is a particularly dusty occupation. (Figures 2.1-2)

There is a small workshop in which a dozen men make asbestos pads or mattresses. These are made from asbestos cloth filled with amosite.

Fig. 2.1 Stripping pipe lagging in a boiler room. Note the dust, mainly amosite asbestos lying on ledges and pipes, and the debris on the deck plates.

The water pipe and ventilation duct insulation has been carried out by sailmakers who apply asbestos cloth over cork, felt, or glass fibre. They have also been responsible for the removal of the old material. Dust is produced when the cloth is torn, and as it quickly blunts sharp knives or scissors the men prefer tearing it.

Sound insulation is applied by joiners, who have produced dust when sawing the friable fibre board, and even more when they rip out old sound insulation.

Shipwrights have been responsible for applying asbestos board to bulkheads and deckheads in machinery spaces, and boilermakers cut and fit asbestos millboard or slabs into boiler casings. These materials used to contain crocidolite according to old Admiralty specifications. Smiths line galley equipment with very friable asbestos millboard and engine fitters cut and fit resin bonded asbestos blocks. Masons have applied asbestos cement over the sprayed asbestos, often in close proximity to the sprayers, and have also used rotary buffing machines to smooth terrazo flooring containing 1% asbestos.

Painters have been responsible for the spraying of asbestos and also for its removal. Both of these operations are extremely hazardous. A mixture of asbestos fibre and cement in a stream of water was sprayed onto the steel deckheads and bulkheads. The resulting layer of asbestos, usually 2"-3" thick was held in place by expanded metal secured by clips to the steel plates, and finally by a layer of asbestos cement. It might be thought that this was not a dusty job because it was wet, but this is far from the truth. The fibre mixture was fed into a hopper and this created a local dust cloud.

In the hopper it was damped and then blown through the nozzles of the hose while water was blown through an adjacent nozzle. Dust was given off when the spray hit the steel surface because the water could not effectively wet the asbestos. The men have told me that if they used too much water in the spray, especially when spraying flat overhead surfaces, then the asbestos formed a wet, soggy mass which became so heavy that it fell off onto the head of the sprayer.

Although most of the fibre was blue, amosite fibre was used occasionally and was disliked because it was more dusty than the blue. Men are described as emerging from the compartments covered from head to foot in dust.

A report prepared by the Advisor on Applied Hygiene to the Medical Director General of the Navy in 1951 on the asbestos spraying process describes the air near the operator's face as being "manifestly heavily charged with asbestos particles" .... "everywhere the space was splattered with asbestos". The report went on to say that "Limpet asbestos spraying as at present carried out in HM Dockyards is always dangerous, and at times intensely so to the health of the workpeople engaged".

Large and variable numbers of labourers have been employed in clearing up asbestos debris and they have been exposed to high concentrations of dust. Firstly the large pieces are bagged and taken out, then the pipes and awkward surfaces are "blown down" with a compressed air hose and the accessible dust is then swept up or sucked up using an industrial vacuum cleaner. This applies to machinery lagging and the sprayed asbestos insulation.

A small number of men are employed in the asbestos stores which issue the materials to the laggers, joiners, etc.

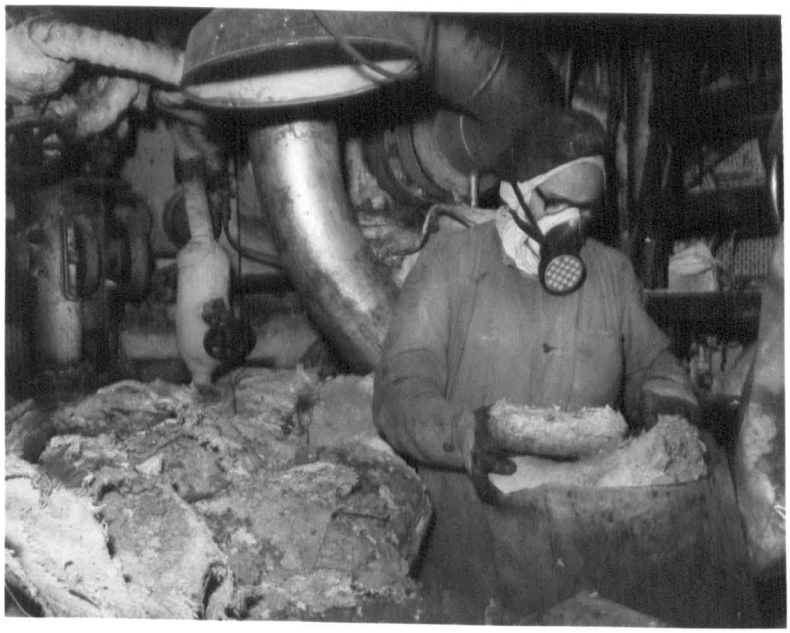


Fig. 2.3 Bagging amosite section debris and asbestos cloth. Note the low headroom. Flash photography does not show the dust.

Fig. 2.4 Two types of industrial vacuum cleaners used to remove the smaller debris.

All these processes, with a few exceptions, take place aboard ships often in small confined spaces filled with machinery and pipes. (Figures 2.3-4). Working conditions are nearly always cramped, and access to the work is by a series of narrow passageways and vertical ladders. All the material has to be carried down by hand, and all the debris is removed in the same way. There is usually no ventilation during the period of the refits, and the modern warship has no port holes so that there is little or no natural ventilation.

The processes and men described have been those directly concerned with asbestos materials, but there have been a great many more at risk. Most of the asbestos is used in machinery compartments and it is in these that many men have been occasionally exposed to asbestos by working near men applying or removing the insulation. It was commonplace to see a lagger, wearing a dust respirator, removing asbestos sections from a pipe and dropping it onto the deck below where it gave off a cloud of asbestos dust into the face of another workman, not wearing a respirator. The men most likely to have been employed in these conditions were boilermakers, engine fitters, shipwrights, electrical fitters, welders, caulkers and various labourers.

The same type of occasional exposure occurred during the asbestos spraying process, or in the removal of sprayed asbestos. Anyone working in the vicinity would be exposed to the dust, but the most likely to have had high exposure would be welders or caulkers concerned with putting up or taking down securing clips, masons, shipwrights, shipfitters, electrical fitters, and again a variety of labourers.

It will be remembered that an official estimate of the number of such men was 2,000, but it is more likely that any of the 6,000 men at work on

the ships may have been exposed to the dust at some time or another. There is considerable movement between employment on the ships and in shops along the dockside and it is reasonable to envisage an even greater number of men with exposure to some asbestos dust over the last 20 years.

PREVENTIVE MEASURES IN DEVONPORT DOCKYARD 1900-1965

From the records of the Medical Department of the Dockyard it is clear that the Navy has always been aware that there is a health hazard associated with the use of asbestos, but that the extent of the risk has not, until recently, been fully realised.

There are records of medical examinations for men working with "silicate cotton and asbestos" from 1912, and these were men of various trades engaged mainly in pipe lagging for machinery and insulating the ship's water supply system. Most of the major machinery lagging was done by contractors, but boilermakers, engine fitters and their assistants removed and replaced small portions of insulation as required. They cut, made and fitted asbestos pads and mattresses, magnesia sections, blue millboard and "plastic" compound in engine rooms and boiler rooms. It is not clear whether or not any form of respiratory protection was issued at this time.

In 1923 the first asbestos mattress making shop was opened and staffed by boilermakers and engine fitters who had experience in making these articles. About twelve men were so employed and they made the mattresses in this shop after measuring the pipes on the ships. On completion of the mattresses they returned to the ships and fitted them on the machinery.

In 1932, following the Report by Merewether and Price on the asbestos industry, a new lagging shop with exhausted booths for mixing, filling and beating the mattresses was created. Technical advice was given by the Factory Inspectorate which at that time belonged to the Home Office. Men working on the ships were issued with dust respirators, but these were simple gauze masks. The mattress shop workers were issued with respirators approved

by the Factory Inspectorate. The shop was subject to the regulations of the Factories Act.

The twelve men working in the mattress shop were also distinguished by being the only men working with asbestos to come under the Home Office Workmen's Compensation Act, Silicosis and Asbestosis Scheme (1931) in which they were required to have initial and periodic medical examinations by members of the appointed Medical Board. It must be remembered that the bulk of the insulating on the ships was still done by insulating contractors, and the Dockyard men of various trades who occasionally applied and removed lagging material were not thought to be exposed to much risk. Some of these men were, in fact, examined periodically by the Dockyard Medical Service, and were sent for chest x-ray if this was thought necessary after physical examination.

In 1943 the Dockyard Medical Service made use of the 35 mm Pulmographic equipment at the Royal Naval Barracks to examine radiologically the chests of those men working in the asbestos mattress shop. Men with abnormal films were called back for investigation, and some were removed from further exposure to asbestos dust. This examination became part of the annual medical examination.

The Ministry of Labour, which took over the Factory Department in 1940, sent a letter to all shipbuilding and shiprepairing yards in August 1945 drawing attention to the increasing use of asbestos insulating materials in ships and the associated hazard. This letter suggested that mattresses should be made under conditions complying with the 1931 Asbestos Industry Regulations and that measures should be taken to improve conditions on ships. It also suggested the supply of respirators to men concerned with asbestos spraying or

working in the same compartment, and also for men fitting or removing dry insulating material containing asbestos. It would seem that these precautions were not fully implemented.

In 1947 the Dockyard formed its own lagging department with men from the asbestos mattress shop, and from the men previously used to doing small insulating jobs. Between 70 and 100 men were employed as laggers over the next few years. At the same time Dockyard employees were sent away to be trained as asbestos sprayers, and they took over this process from the contractors. Seventy-three men were trained between 1947-1960. All these men came into the Dockyard scheme for medical examination including chest x-ray.

At this time there does not appear to have been any formal code of practice for laggers, or sprayers, but they were all issued with Siebe Gorman Mark IV respirators. The conditions created by the sprayers were so obnoxious that most other trades refused to work in the same compartments, but some welders, masons, and various labourers had to work in close proximity to them. Occasional exposure to asbestos from lagging or stripping of the insulating materials was not thought to be hazardous and no methods of segregating the work were employed.

By 1949 the Dockyard had a code of practice for asbestos spraying and this included initial and monthly medical examinations, with 6 monthly chest x-ray. Siebe Gorman Mark IV dust respirators and eye goggles were provided and cotton overalls changed weekly. Arrangements were made for some ventilation in the compartments. The asbestos was to be pre-damped and debris to be cleaned up regularly. As far as practicable other tradesmen were not allowed in the vicinity of spraying operations, and those that were allowed in

were to wear respirators.

In 1951 the Advisor in Applied Hygiene to the Royal Navy made his report to the Medical Director General on the asbestos spraying process together with recommendations for a code of practice. He showed that the process was very hazardous and his recommendations were similar in principle to the regulations then in force. It was agreed that medical examinations should be carried out initially, six monthly, and annually to include a chest x-ray. These and more detailed requirements were published as Instructions to each Dockyard.

In 1954 the Senior Medical Officer in Devonport Dockyard decided to investigate whether or not exposure to dusts, including asbestos, had had any effect on men working on ships under refit, and 93 men were examined by the local chest clinic. There was no suggestion of any type of pneumoconiosis, and further precautions did not seem to be indicated.

The joiners using acoustic asbestos fibre board were later thought to be at risk, but after a cursory investigation in 1957 they were thought not to have sufficient exposure to the dust to constitute any risk of asbestosis.

A dissertation presented for the Diploma in Public Health at the London School of Hygiene in 1959 by J.D. Walters, a Naval Medical Officer at Portsmouth Dockyard gave a clear account of the situation with regard to the use of asbestos in the Dockyards at that time. He drew attention to the fact that men other than the laggers and sprayers were at risk, that stripping asbestos was often more hazardous than its application, and outlined methods of preventing the disease by substitution, damping, segregation and better personal protection. He also advocated the setting up of an asbestos workers register, with an employees record card which could be taken from one employer to another. He drew attention to the difficulty

of diagnosis of the disease.

In 1960 the Senior Medical Officer of the Dockyard again decided that asbestos workers were at risk, and 4, out of the 88 laggers x-rayed, were thought by the Naval Radiologist to have films suggesting asbestosis. These men were investigated at the Royal Naval Hospital, Plymouth, where the findings were confirmed and their cases were discussed with the Medical Inspector of Factories for the area and the Pneumoconiosis Medical Panel. One of the four men was accepted by the Panel as suffering from asbestosis, but as far as we can tell from the records the other three did not attend for examination.

Protective measures were reviewed at this time and because it seemed impracticable to get the men to wear air fed hoods, or even dust respirators properly, the suggestion was made that laggers should only be employed on this work for up to 5 years in an attempt to limit the exposure. This was not implemented. The men working with asbestos were issued with Siebe Gorman Mark IV dust respirators and they were later replaced with Micro-filter respirators. These had a plastic mouthpiece which did not fit well to the face and were replaced by Siebe Gorman Mark VIII dust respirators which had rubber face pieces. Unfortunately many of the Mark VIII respirators were issued with fume cartridges in place of dust filters and provided little protection against dust.

The situation in 1965 was that all the laggers were examined annually at the Dockyard Medical Centre, including 70 mm chest x-ray, and they were issued with respirators. The twelve laggers who worked in the mattress shop were also examined every two years by the Pneumoconiosis Medical Panel.

The sprayers by this time had all reverted to other trades, and as the process was not in use the men were not examined. All other Dockyard workers aboard ships were unprotected and their exposure continued to be varied and intermittent.

The Factories Act 1961, and the Asbestos Regulations 1931 applied to the asbestos mattress shop. The Shipbuilding and Shiprepairing Regulations 1960 made general provision, in Regulation 39, for the removal of dusts and fumes aboard ships. Regulation 76 required the provision of approved breathing apparatus for men exposed to dust while spraying asbestos, breaking down asbestos lagging, or cutting asbestos with a portable power saw. These regulations were applied to work in ships and on the docksides.

It can be seen that at this stage the statutory regulations were insufficient to do more than give some degree of protection to mattress makers, sprayers, and men stripping lagging. The other users of asbestos materials and the large number of men working in the ships were outside the regulations.

The foregoing account attempts to describe the processes, materials and men associated with asbestos in Naval Dockyards in general, and the Devonport Yard in particular. It is easy to be critical of the events of the past, but I am convinced that the Dockyard authorities did as much as, if not more than any other employer in the shipbuilding industry to protect the men according to their current knowledge. The absence of demonstrable disease was the main reason why energetic preventive measures were not taken earlier.

These were the conditions existing up until 1965 when, as the direct result of the investigations now being described, measures were introduced to attempt to control the hazard.

PROTECTIVE MEASURES DEVELOPED IN DEVONPORT DOCKYARD SINCE 1965

The representations made to the Medical Director General (Naval) in 1965 resulted in the initiation of the present survey into the asbestos hazard and when the extent of the problem was realised the facts were placed before the Dockyard authorities.

At first, Production Managers were sceptical of the extent of the hazard. Few cases of asbestosis had been confirmed, and no deaths were known to have been directly attributed to exposure to asbestos. However, the x-ray films of the men forming the main study population (to be described later) showed that about 40% of them were abnormal in some respect and this helped to emphasize the need for energetic preventive measures.

The two main users of asbestos were the Engineering Department which used vast quantities for machinery insulation, and the Constructive Department which was busy replacing sprayed crocidolite asbestos with glass fibre and also used a lot of asbestos for sound insulation and for chilled water systems.

The first approach was to look for substitute materials and the Constructive Department immediately stopped using asbestos cloth for cold and hot water pipes, ventilation trunking and chilled water lines. Soft fibre board previously extensively used for sound insulation was replaced by glass fibre and covered with perforated asbestos cement board which did not give off asbestos dust.

The Engineering Department was faced with more difficult problems of substitution, but the remaining stocks of amosite sections were abandoned in favour of calcium silicate sections, which contained much less asbestos, and a variety of other alternative materials were tried.

Work is now progressing, under the supervision of the Navy Department Materials Section, into the use of alternative materials for insulating machinery in HM Ships and it is hoped that the amount of asbestos contained in materials liable to produce airborne dust will either be eliminated or reduced to a very small amount. Two ships are being insulated with pipe sections containing only 5% asbestos as an inner layer and overlaid with asbestos-free magnesia section. These are then covered with glass fibre cloth. For turbine insulation an asbestos-free ceramic insulation has been tried in the form of a spray, and this is now undergoing sea trials. It may be possible to replace the asbestos mattress with one made from glass fibre cloth and filled with mineral wool. Asbestos cloth containing a dust suppressant has been developed by the asbestos industry and is a big improvement on the old cloth.

All other uses for asbestos materials are being similarly studied and alternative materials supplied where possible.

The reason why substitution is the main form of tackling the problem in shipbuilding is fairly obvious from the previous review of the nature of the work. No two ships are the same, the work varies from hour to hour and it is virtually impossible to apply to work in these ships the standard of dust control that may be possible in factories. This will become more readily apparent in the presentation of the dust sampling data.

It was obvious that not only would substitution be difficult, but it would take time to implement, and in the meantime, vast quantities of asbestos materials already in the ships had to be manipulated during their refitting periods. In order to avoid the haphazard asbestos exposure of the past new working routines would have to be introduced.

Broadly, the methods advised were, segregation of the processes, damping the materials to reduce dust levels, better ventilation systems, and better personal protection for the men. All of these were said to be impracticable, but with the active co-operation of management, unions, and men they were all tried with various degrees of success. It was argued that all these measures would increase the time and hence the cost of the work. The damping methods certainly did, but by suitable planning it was found that time was sometimes saved by segregating both the stripping and relagging procedures, as other trades did not then interfere with this work.

Damping methods failed. Low pressure water sprays and probes used to penetrate and infuse the lagging were of very limited value (Figures 2.5 and 6). They did not dampen the material very successfully and the probe method was especially tedious and time consuming. Other disadvantages were discovered. The water increased the weight of debris which all had to be manhandled out of the ship, sometimes up 8 vertical ladders. The wet material also formed a soggy mass underfoot which could not be cleaned with vacuum cleaners and which later dried only to be powdered into dust by other workmen later in the refit. It was decided for these reasons to abandon wetting methods and to rely on better personal protection and efficient cleaning of the compartments.

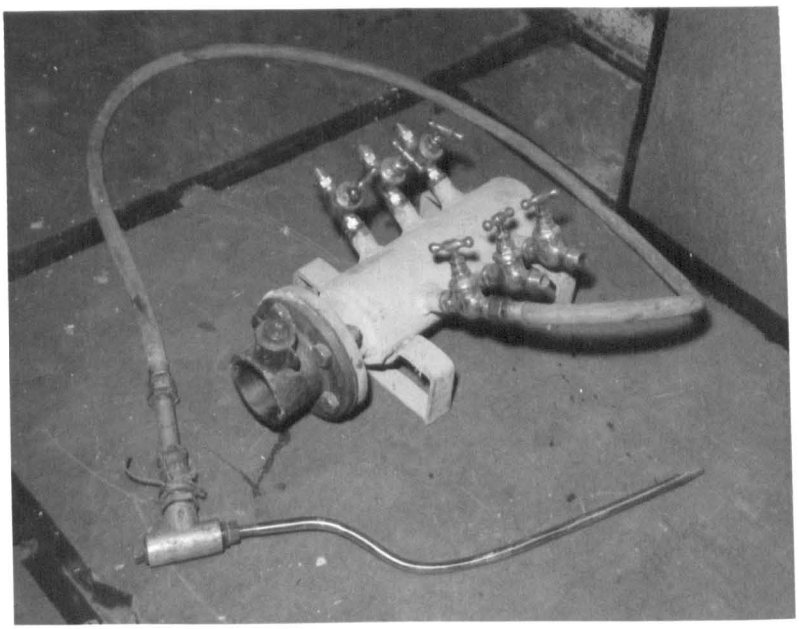


Fig. 2.5 One of the probes designed to penetrate and infuse the sprayed asbestos on deckheads.

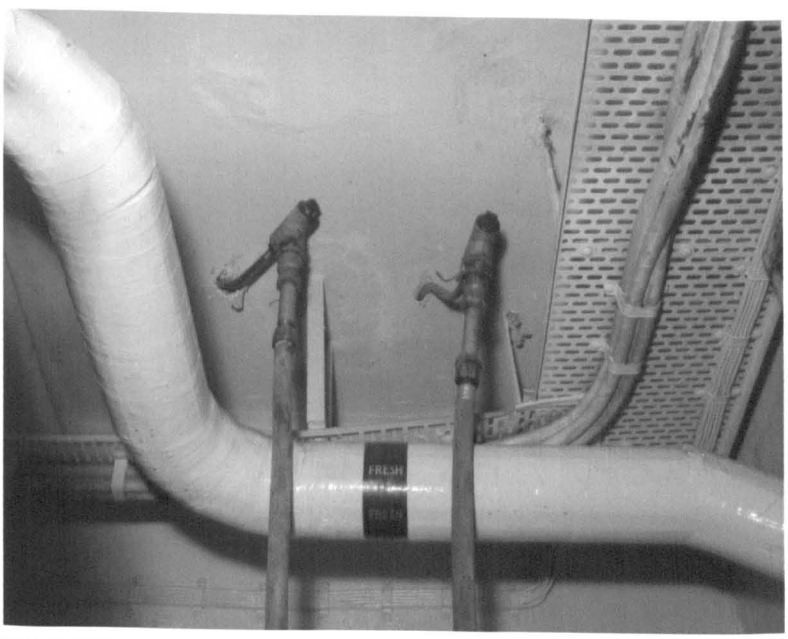


Fig. 2.6 Probes in position, driven into the sprayed asbestos.

The refit of an aircraft carrier in which all of the machinery insulation in the four engine rooms, four boiler rooms, and most of the environmental sprayed crocidolite asbestos was to be removed, seemed an ideal opportunity to put the theories into practice.

The main machinery spaces were isolated and were stripped of all their insulation before the ship had finished de-storing and came into Dockyard hands. Notices were displayed prohibiting entry to all except the men concerned who were supplied with Siebe Gorman Mark VIII respirators. Provision was made on the ship for the men to have changing and shower facilities so that there was no danger of contamination of their hair or outdoor clothes. The debris was placed in polythene sacks, sealed and placed in a barge to be dumped at sea. Unfortunately, the bags floated, so they were replaced by paper bags which were well soaked with water before dumping and these sank successfully. Before other workmen were allowed into the machinery spaces the compartments were carefully cleaned with industrial vacuum cleaners and the previous problem of large quantities of asbestos debris remaining to be disturbed was avoided. The same precautions were taken for the numerous smaller machinery spaces throughout the ship.

The removal of most of the environmental insulation was the other major problem to be tackled. This involved the removal of more than 750,000 square feet of sprayed crocidolite asbestos which was between 2 and 4 inches thick.

Wetting methods had previously been shown to be impracticable so that again segregation of the work and improved personal protection were the

principal methods used to deal with the problem.

Because of the need for escape routes in case of fire the main hatches and bulkhead doors could not be locked, so the parts of the ship in which this work was to be done were sealed with hardboard panels.

A work schedule was drawn up and some 200 men worked day and night shifts to complete the task which occupied them for nine months. All other men were kept out of the "contaminated" areas until they had been cleaned, and if men had to enter for essential repairs to lighting circuits then they had to wear the same protective clothing as the asbestos strippers.

The men were issued with a complete change of clothes, vest, shirt, shorts, socks and boots, an impervious PVC or a rubberized canvas overall, and a PVC air line respirator which was supplied with air from the Dockyard compressed air line through an oil and water filter. (Figures 2.7, 8 and 9). Changing facilities were arranged on the ship which included showers through which the men had to pass on the way back from the contaminated areas. (Figures 2.10 and 11). Supervisors, or short term visitors such as Safety Officers, Constructors or Engineers were required to wear the same overall, but had a full face piece dust respirator fitted with a PVC hood. (Figure 2.12).

There were obvious "teething troubles" with the equipment such as misting of the face piece of the air hood, excessive sweating in the suit, and long delays during the changing periods. These problems were solved by adjusting the air flow of the hood, by supplying orange or lemon juice and salt tablets on demand and by altering the working shifts.

Fig. 2.7 Clothes issued to men removing sprayed crocidolite asbestos.

Fig. 2.8 Tools, and filter unit for supplying air to men removing sprayed crocidolite asbestos.

Fig. 2.13 One half of hangar deck of an aircraft carrier. The undersurface of the deckhead was coated with 2"-3" of sprayed crocidolite.

Fig. 2.14 An early attempt at dust extraction. The dust laden air passed through a spraying compartment. The volume of air moved was insufficient to have any appreciable effect on working conditions.

Fig. 2.17 Left bag contains blue crocidolite debris, right bag shows white amosite cement and expanded metal retaining mesh.

Despite the obvious discomfort of the equipment, and the very arduous nature of the work there were no serious complaints from the men, and no detectable adverse effects on their health. Transient trouble with skin rashes on the faces of supervisors and others wearing the full face piece respirators were solved by cleaning the face pieces with soap and water instead of disinfectant solution. By and large the opinion of the men was that at last they were having reasonable protection from the dust.

The amount of work involved was enormous. The whole of the under-surface of the hangar deck, (Figure 2.13) nearly all the mess decks, officers' quarters and other spaces were covered with sprayed asbestos which was to be removed. To do this, wooden staging was erected within four feet of the overhead deck and the planks were covered with polythene sheeting to attempt to contain the debris. Half the vast space of the hangar was tackled first, the other half curtained off by the fire curtain and turned into temporary storerooms, workshops and changing rooms. This proved not to be very safe as will be shown later in the dust sampling survey.

The men doing the stripping attacked the material, which was coated with asbestos cement and held onto the deckhead by expanded metal and clips, with small crow bars, chisels and hammers. (Figure 2.15). The atmosphere soon became thick with dust and it was disturbing to realise that until now these conditions had persisted for half of a long refit with no attempt to contain the dust or to protect men other than those actually involved in the work. Attempts were made to increase the ventilation by using portable dust extracting units, but these were not successful. (Figure 2.14).

The remaining spaces were of varying sizes and shapes, some large, some very small and confined, but all difficult of access and requiring a great

deal of labour to remove the debris from the ship. This was done by a special gang of men wearing the same protective overalls and air line respirators while sweeping up and bagging the debris, but Mark VIII respirators while carrying it out to the barge. (Figure 2.16). The paper bags containing the debris were filled with water before being dropped into the barge for disposal at sea. (Figure 2.17).

After most of the debris had been removed the area was brushed, cleaned with industrial vacuum cleaners, and finally with damp cloths. Then paint sprayers, suitably protected, gave the steel plates a covering of primer paint in order to seal any remaining asbestos fibre.

Similar precautions were taken for the removal of asbestos acoustic boards which also proved to be extremely dusty. This material was fibre board $1\frac{1}{2}$" thick held in position by wooden battens and covered with wooden boards, and had been in place for about 20 years. The amount of dust caused by ripping out the wooden boards, and then the underlying asbestos is indescribable, but the dust sampling showed that most of it was not asbestos and that it was of large particle size, so that it was perhaps not quite such a dangerous occupation as it looked.

The experience gained from these procedures has been applied to all other asbestos processes in the Dockyard. Where possible the work is isolated and the men protected. Where there are heavy dust concentrations, impervious suits and air line respirators are used, and for lower dust concentrations nylon overalls with approved dust respirators. Positive pressure power respirators were also tried at this time and found to be very acceptable to the men. They are comfortable to wear and have no trailing air hoses which are a hindrance in confined machinery spaces.

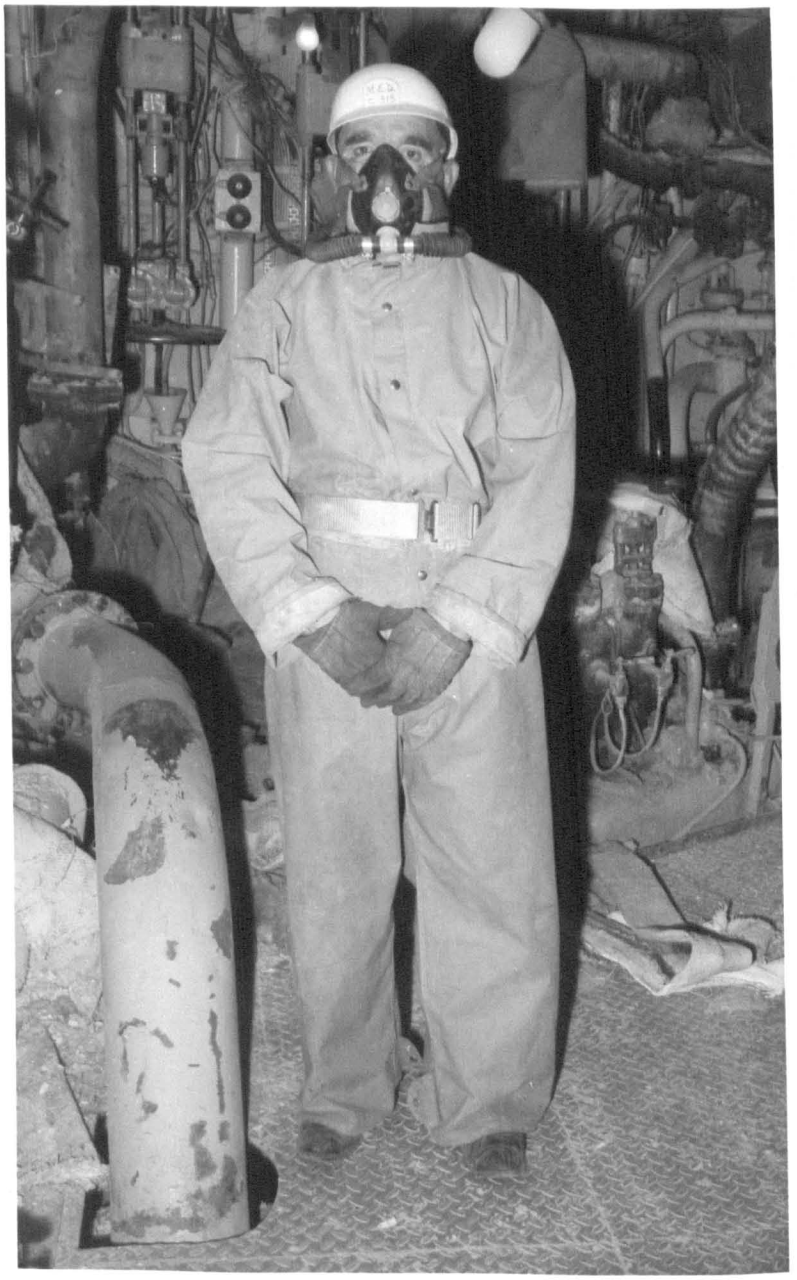


Fig. 2.18 Lagger stripping amosite pipe lagging wearing impervious rubber suit and positive pressure power respirator.

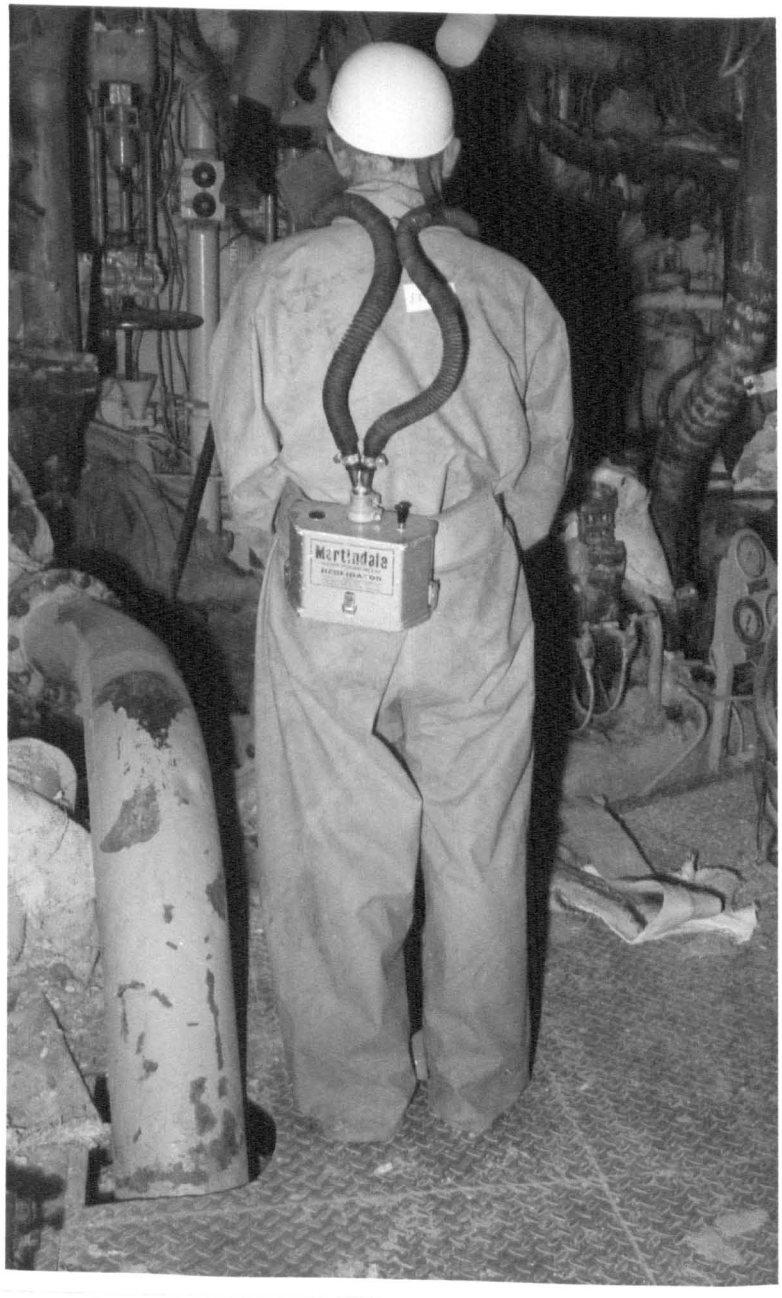


Fig. 2.19 Back view of the same man showing the power unit. The air is drawn through a filter at the base of the unit and delivered to the face piece by the rubber tubes.

Fig. 2.20 Sewing an asbestos mattress in the old mattress making shop. Note clothing hanging in shop and piles of completed pads on which dust will settle.

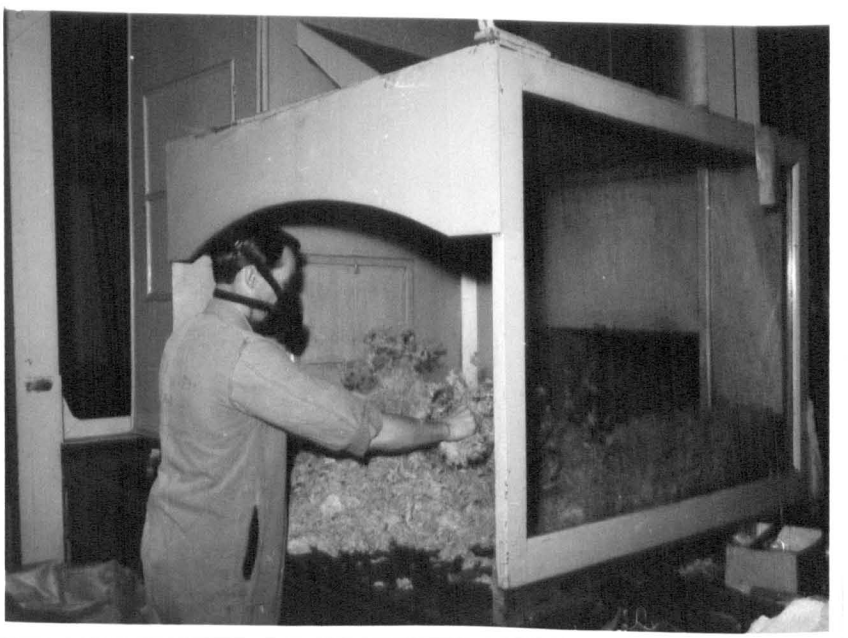


Fig. 2.21 Old mattress shop. Filling booth dating from 1933. Poor extraction and very big opening meant very little dust was taken away from the man. Note bag of fibre alongside him.

Fig. 2.22 Newly constructed asbestos mattress shop showing improved exhaust ventilated filling cabinets.

Fig. 2.23 Sewing mattresses in new shop. Dust suppressed asbestos cloth filled with mineral rockwool. Glass fibre cloth is replacing asbestos cloth. Note men only wear respirators while at the filling cabinets.

Recent modifications have made this respirator even more attractive for use in high dust concentrations. (Figures 2.18 and 19).

The increased number of respirators now being used called for expert care and maintenance and this is now undertaken at a central unit which cleans, maintains, repairs and issues all types of respirators.

At the same time improved accommodation for the asbestos workers was recommended and this has now been completed. It consists of an improved asbestos mattress shop with better exhaust booths for filling and beating the mattresses, and a workshop where polyurethane lagging for glands are prefabricated instead of asbestos cement and cloth lagging. (Figures 2.20, 21 and 22). The changing accommodation is palatial. The men enter, leave their outdoor clothes in lockers and are issued with a complete change of clothes, nylon overall and respirator. On return from the ship they enter through another door, leaving their overall and respirator for cleaning, and then proceed to shower before dressing in their outdoor clothes. There is a separate rest and dining room, and a laundry.

All these measures are the result of trials under working conditions and have been improved or discarded according to their effectiveness and practicability. As conditions or materials change so will the extent of protective methods. The new Asbestos Regulations which are to be laid before Parliament in 1969 will apply to all the users of asbestos in the Dockyards, and not only to the mattress makers. The preventive methods adopted by Naval Dockyards are in accordance with the proposed regulations and they have already been proved to be practicable in this complex industry.

This does not mean to say that the hazard has yet been eliminated. I consider that with the progress now made in improved working conditions, and the work proceeding on alternative materials or on materials which do not give off so much dust, then we should expect to find in 10-15 years time a marked decrease in the number of men who may be affected by the dust.

A SURVEY OF ASBESTOS DUST CONCENTRATIONS IN DEVONPORT DOCKYARD

ASBESTOS DUST CONCENTRATIONS FOUND IN INSULATING AND OTHER PROCESSES AT HM DOCKYARD, DEVONPORT

REVIEW OF THE LITERATURE

There are few published reports describing asbestos dust concentrations occurring in the ship building and ship repairing industry. In 1946 Fleischer and his colleagues carried out a survey of four United States Dockyards which built naval vessels. They described the materials and processes, gave the results of a dust sampling study and presented the findings of radiological examinations of shipyard pipe coverers.

Much of the material contained more than 95% amosite with some chrysotile asbestos. The processes were concerned with machinery and pipe insulation in ships, and mattress making and prefabrication of the insulating materials in the shops. Dust samples were taken with a Konimeter and expressed as millions of particles per cubic foot of air (mppcf).

The dust concentrations showed that bandsaw cutting of asbestos sections, mixing asbestos cement, and installation of asbestos material aboard ship were the dustiest operations. The report recommended that exhaust ventilation should be applied to these processes. It was noted that it was very rare to see a man wearing a dust respirator. The changing nature of the work from day to day, and ship to ship, made it very difficult to obtain an accurate picture of the amount of dust any one man might have been exposed to over a period of years. For these reasons the authors thought that it was virtually impossible to set threshold limits for asbestos exposure in the ship building industry.

There was a very wide range of dust concentrations and much of the dust seen in the samples collected by the Konimeter was not asbestos. The

average concentrations in the four Yards were:-

Shop work	Total dust	19.3 mppcf	Asbestos fibre	1 mppcf
Ship work	Total dust	82.5 mppcf	Asbestos fibre	1 mppcf

One of the four Yards was surveyed again in 1966 by Murphy and Ferris and comparative values for average dust concentrations in this Yard as given in the two reports are:-

Fleischer et al

1946	Shop work	Total dust 32.0 mppcf	Asbestos fibre 2.6 mppcf
	Ship work	Total dust 49.2 mppcf	Asbestos fibre 1.1 mppcf

Murphy & Ferris

1966	Shop work	Total dust 25.8 mppcf	Asbestos fibre 6.9 mppcf
	Ship work	Total dust 26.2 mppcf	Asbestos fibre 0.02 mppcf

In 1964 a study by Marr described the conditions in Long Beach Naval Shipyard. He described the materials in use, and drew attention to the large quantities of amosite asbestos that had been used, and to the presence of diatomaceous earth, magnesia, glass fibre and other dusty substances used in the insulating materials which might complicate dust sampling and the exposure hazard. He mentioned the widespread removal of insulating material that had occurred during ship overhaul and modernisation since 1945. Dust samples were taken with a Bausch and Lomb dust counter and the results show a wide range of concentrations. Fibre counts were reported as being only a "trace" for all the operations except for the removal of amosite asbestos blankets (0.5-8.0 mppcf), installation of 85% magnesia with 15% amosite asbestos segments (0.1-1.8 mppcf), and the removal of these sections (trace - 1.2 mppcf). Fibres between 3-60 microns long were counted. Particle counts for all processes ranged between 0.1-10 mppcf for particles 2-10 microns diameter.

In his report Marr points out that the men were exposed for short periods to high concentrations of asbestos dust during the removal of old insulating material, and that there were no established figures for a maximum allowable concentration of asbestos fibre for these short massive exposures, or for pipe covering in general. He mentions that the use of X-ray examination of men exposed to asbestos can be misleading if it is not realised that asbestosis usually takes at least seven years to develop, and that it is important to examine those with longest exposure to asbestos. He concluded that pipe covering in ship overhaul and repair was a hazardous trade and that men should wear a respirator when exposed to dry asbestos insulating material.

Ferris (personal communication) using a Bausch and Lomb dust counter found particle counts ranging from 7-130 mppcf during the removal of amosite asbestos in boiler rooms, and from 10-112 mppcf during the removal of magnesia block and amosite insulation. The size of the particles counted is not given in this communication.

Fleischer and his colleagues deduced from their survey that the work involving asbestos products in ship building was not hazardous because only 3 of the 1074 men examined showed signs of asbestosis, and all three of them had worked for more than 20 years. Localized high dust concentrations occurred intermittently and men were not constantly exposed to such high concentrations. This is probably true, but, as later reviewers have pointed out, the most likely explanation for such a low number of affected men is probably that few of them had been exposed to the dust for long enough.

Marr records that 5 men out of 60-80 insulators had asbestosis, but

this survey was not reported in sufficient detail to give any true impression of the incidence of the disease. The results from the dust samples showed that tearing out old insulation was very dusty work, and that this type of work had increased since the end of the 1939-45 war. Murphy and Ferris confirm these high dust levels during tearing out processes, and also that with the increase in number of men who had been exposed to the dust for more than 20 years, there was an increase in the number of men who developed asbestosis.

Balzer (1968) and Balzer and Cooper (1968) describe the workmen, products and processes associated with insulation work in the San Fransisco Bay area. Sixty of 380 insulators worked in marine construction and repair. The reports mentioned that 80% of the insulating materials used in marine work contained asbestos and the men worked with asbestos materials for 45% of their working time.

Both papers explain the difficulty of making accurate assessments of asbestos exposure in the insulating trade, but show the wide range of dust concentrations found during various processes. Dust samples were taken with a midget impinger; grains and fibres were counted under a light field microscope at x 100 magnification, and the results expressed in mppcf. Membrane filter samples were also taken and all fibres with diameter less than 3.5 microns were counted under phase contrast illumination at x 430 magnification.

The processes were divided into prefabrication; application; finishing; tearing out; mixing; and general samples. Prefabrication, involving the use of hand and power saws (6.5 mppcf); mixing (9.1 mppcf); and tearing out (5.2 mppcf) gave the highest mean values with the midget

impinger. The overall range of values was 0.6-28.8 mppcf. The membrane filter results showed that tearing out (8.5 f/c.c.) and prefabrication (8.5 f/c.c.) gave the highest mean concentrations, with application (6.4 f/c.c.), general cleaning up and carrying material (4.8 f/c.c.) and mixing (2.6 f/c.c.). The overall range of counts was 0.1-61.6 f/c.c.

We are not told in these reports which of the samples were taken in the factories, buildings, or ships, and it is important to remember that these are overall mean values if they are to be compared with the values to be given in the present report.

The only other report of dust concentrations occurring in insulating work is that of Leathart and Sanderson (1963). In this paper the varied work of an insulator is described together with a brief account of the materials and processes. Dust samples were taken with a thermal precipitator during the insulation of pipe work for a hospital heating system. Fibres between 5 and 50 microns long were counted. Mixing asbestos cement in a bucket gave a count of more than 250 f/c.c. and sweeping up stripped asbestos debris produced dust concentrations of between 2 and 23 f/c.c.

Leathart and Sanderson suggest that high counts were not likely from the other processes undertaken by the laggers in the work at that hospital, but suggest that worse conditions might exist during similar work in ships or other buildings and that this required further investigation.

All these reports mention the difficulty of estimating the overall exposure to asbestos for insulating workers, and most of them make reference to the Threshold Limit Value (TLV) of 5 mppcf which was recommended by the American Conference of Governmental Industrial Hygienists (ACGIH) in 1946. This was based on the work of Dreessen et al (1938).

Schall (1965) has written an appreciation of the study carried out by Dreessen and his colleagues. Amongst other pertinent comments he drew attention to the fact that the value of 5 mppcf included all fibrous and non-fibrous material, asbestos or not, and that particulate matter predominated in the samples. He also noted the very wide range of dust concentrations, and suggested that there was some biological evidence to suggest that peak exposures were more important than constant overall background exposure since huge retention of particles might take place on those occasions when the defence mechanisms were overwhelmed.

In Dreessen's study 333 of 511 men had worked with asbestos for less than 5 years, and only 66 of 511 had worked for more than 10 years. Lynch and Ayer (1968) point out that prior to Dreessen's study 150 workers out of less than 600 had been discharged from work as suspected cases of asbestosis. It is inferred that if the study had included more men who had longer exposure to asbestos, then the incidence of the disease would have been greater, and this would probably had resulted in a lower recommended Threshold Limit Value. However Dreessen et al, did suggest that the TLV was a tentative value until better data were available.

Lynch and Ayer (1966) give details of dust concentrations in American textile factories before and after the introduction of dust control measures. From the results we can see the dramatic improvements made towards controlling the dust hazard, and how bad conditions must have been before their introduction.

Before further discussion of the TLV for asbestos, and its application to insulating processes, it is important to remember that the TLV relates to experience gained in American textile factories using chrysotile asbestos.

Samples were taken with the midget impinger, all particles were counted using a light field microscope at x 100 magnification.

The different types of asbestos fibre have different physical characteristics which influence their collection by sampling instruments, and also their penetration and retention in the lung tissues (Timbrell 1969 personal communication). Also each type of asbestos probably has a different capacity for producing fibrosis of the lung.

Furthermore the various materials used for insulating work contain not only different amounts, but different varieties of asbestos, so that the composition of the dust clouds produced in this type of work is extremely variable.

For these reasons alone it would be unwise to attempt to apply the present TLV to working conditions in the insulating trade. It is even more difficult if different sampling techniques are employed to estimate the dust concentrations.

The impinger has the disadvantage of being inefficient at collecting fibres, and under the light field microscope few fibres are seen at the usual x 100 magnification. For these reasons Dreessen et al counted all particles to give a measure of overall dustiness.

The values given in the reports by Fleischer et al; Marr; and Murphy and Ferris are useful in showing that high dust concentrations occur in some of the insulating processes in ships, but cannot strictly be compared with the present TLV for the reasons given earlier. Ferris (personal communication) has suggested that a TLV of 1 mppcf should be recommended for shipyard insulation work.

The discussion so far has related to the TLV based on total particle counts. The British Asbestos Industry has developed a method for determining the number of fibres per cubic centimetre of air using membrane filters. Fibres are counted between 5-100 microns long and with an aspect ratio of more than 3:1, at x 430 magnification under phase contrast illumination. (Holmes 1965). There has been no official TLV, but an informal level of 4 f/c.c. has been regarded as the upper acceptable level of dust concentrations for planning dust control measures in textile factories using chrysotile asbestos, and in a factory making insulating sections and asbestos sheets from amosite fibre (Roach 1965). This method has obvious attractions for use in the insulating processes because of the widely different asbestos content of the materials.

A comparison of the performance of the midget impinger and membrane filter technique of measuring dust concentrations in textile mills using chrysotile asbestos has been reported by Ayer, Lynch and Fanney (1965). They suggest that for the processes from which their samples were taken concentrations near the TLV of 5 mppcf with the impinger would be roughly equivalent to 50 fibres/c.c. if all fibres were counted using the membrane filter method.

Roach (1965), in his comparative study of dust sampling techniques in a factory processing amosite fibre, shows that for all visible particles the mean value for the midget impinger samples was 1.1 mppcf and the corresponding membrane filter count 21 particles/c.c. For airborne fibres the midget impinger mean value was 0.11 m fibres pcf and the membrane filter count 13 f/c.c. He explains that fibres are not efficiently collected by the impinger.

Roach also compared the performance of gravimetric samplers, and suggests that for the dust clouds from which his samples were taken the TLV of 5 mppcf appeared to correspond to about 1.5 mgm/m^3 of "respirable" dust. He concluded that measurement of mass concentration was simpler, quicker and more accurate than particle or fibre counting methods.

Gravimetric samples would be satisfactory for monitoring the work environment where roughly the same type, and amount of asbestos is subjected to the same process for long periods. For shipyard insulation in which processes and materials are continuously changing, then gravimetric samples would be expected to be of little value in the estimation of asbestos exposure.

In 1968 the British Occupational Hygiene Society sub committee on asbestos published its report on Hygienic Standards for Chrysotile Asbestos Dust. The report suggests that there may be a small risk to health if there is any airborne chrysotile dust in the work environment, but that it would be proper to reduce the risk of contracting the earliest signs of asbestosis to 1% of those with a life time exposure to the dust. To achieve this the report suggests that exposure to chrysotile dust should be limited to 100 fibre years per cm^3. That is 2 fibres/c.c. for 50 years; 10 fibres per c.c. for 10 years. Fibres were defined as particles over 5 microns long with an aspect ratio 3:1 estimated by the standard membrane filter technique. These proposals are described as the best obtainable from existing data. They were based on the study of two groups of workers in a textile factory processing chrysotile asbestos and which is described in the report.

Different dust sampling instruments which could be used for monitoring the working environment are described, and sampling procedures suggested for

evaluating the dust category for each process. It is suggested that these categories should be used to specify the type of protection to be provided for the worker.

An appendix to the Draft Asbestos Regulations which were to have been laid before Parliament in 1968 has been published by the Factory Inspectorate of the Department of Employment and Productivity. The appendix suggests provisional Threshold limits for asbestos for the guidance of HM Factory Inspectors and the users of asbestos, and the regulations will apply to the insulation trade as well as textile factories and other processes involving asbestos.

For chrysotile, amosite and anthophyllyte asbestos the proposed TLV is 2 fibres/c.c. (0.1 mgm/m^3 asbestos), and for crocidolite 0.2 fibres/c.c. (0.01 mgm/m^3 asbestos). The severe limit proposed for crocidolite is because of the association between this type of asbestos and mesothelioma of the pleura and peritoneum.

The situation relating to hygienic standards for the different types of asbestos appears to be confused. Most of the data refer to the use of chrysotile asbestos in textile factories. Very little information exists about dust concentrations and the incidence of asbestosis among workpeople exposed to other forms of asbestos or among insulation workers exposed to all forms of asbestos.

The TLV proposed by the ACGIH based on the 1938 report brought about big improvements in dust control, and must have reduced the incidence of asbestosis in American textile workers. It would appear that this level of 5 mppcf is probably too high to reduce the risk of asbestosis to an acceptable minimum.

It is not clear whether or not the TLV can equally be applied to all forms of asbestos, or to other processes. From existing studies it would seem that dust concentrations in shipyard insulation work have not yet been reduced to the TLV of 5 mppcf.

The proposed provisional TLV published by the British Factory Inspectorate is an attempt to control dust concentrations in all processes involving the use of asbestos to a level at which the risk of developing asbestosis is very small.

Apart from the provisional TLV for crocidolite, proposed by the Factory Inspectorate, all the threshold values apply to the prevention of asbestosis and not to malignant disease associated with asbestos. It remains to be seen, when industry has complied with these proposals, whether they will prove to be effective in preventing malignant tumours due to asbestos.

To provide a clearer picture of the dust concentrations which occur in British Naval Dockyards it was decided to carry out a dust sampling survey, and the results of this work are now presented.

DUST SAMPLING SURVEY

The asbestos materials and the many processes involving them have been described. This is an account of the dust concentrations occurring in most of those processes in HM Dockyard, Devonport.

Methods

A gravimetric method was used to give an overall picture of dust concentrations, but as this method would collect all the dusts airborne and not allow them to be differentiated it was decided also to use membrane filter methods as these enable the estimation to be restricted to the concentrations of asbestos fibre.

Six M.R.E. Casella Type 113A samplers fitted with horizontal elutriators which only allow the "respirable" fraction of the dust to reach the collecting filter were used for the gravimetric estimations. Three of these samplers were fitted with a by-pass for the elutriator in order to collect "total" dust.

The performance of the horizontal elutriator in sampling the "respirable" fraction of asbestos dust has been studied by Higgins (1967). While it was agreed that the use of the M.R.E. samplers may not result in the true measurement of the "respirable" asbestos, at least it gives a comparable measure of the amount of "respirable" dust produced in the various processes.

The samples were collected on Whatman's 5.5 cm GF/A glass fibre filters and weighed to within 0.01 mgm.

Long period samples for fibre counts were taken using four Hunt personal samplers (Hunt and Ellison 1963) which run at 11.3 ml/min, the dust being collected on 20 mm Millipore Type G.A. membrane filters, pore size 0.45. Austen Dymas pumps sampling at 200 ml/min, and Draeger hand pumps

taking 100 ml in about 4.3 seconds were used with 25 mm Millipore Type G.A. filters pore size 0.45 to take shorter samples.

The method of fixing the sample, and clearing the membrane was that described by Holmes (1965) as was the counting technique. The samples were counted using phase contrast microscopy (magnification X 400), and one technician was responsible for taking the samples and their evaluation. Fibres were counted between 5-100 micron in length and with an aspect (length to breadth) ratio of 3:1 or over.

The sampling strategy was planned in an attempt to give a rough idea of the dust concentrations likely to have been present in the ships over the last twenty years. The use of different materials as the result of revised design specification, as well as substitution to reduce the dust hazard, made it necessary to take the bulk of these samples in ships containing the old materials in order to give some idea of past conditions. The opportunity to do this occurred in an aircraft carrier and a cruiser in 1967. The samples of other processes, especially the rebuilding processes have been taken in various destroyers and frigates, as well as the larger vessels in order to give an idea of present working conditions.

Long period gravimetric samples were taken during the whole working shifts. The samplers were placed at head height in positions where the work was taking place, and also in another convenient position to give an estimate of the overall dust concentration of the compartment. Wherever possible the long running Hunt samplers were placed alongside the gravimetric samplers in order to have an estimate of the fibre content of the dust. These samplers were also worn by men undertaking certain jobs to give an estimate of personal exposure.

Many samples were taken using Dräger hand pumps to obtain 200-2,000 ml samples. The disadvantages of small, quick samples are recognised, but it is considered that these are of value as a practicable method of indicating the fluctuation in the local dust concentrations of various processes. The samples were taken at specified points in the general environment as well as in the breathing zones of the operators. These small samples are also useful in showing the change in dust concentrations at varying distances or the rate of change after work had stopped. These methods now form the basis for dust monitoring equipment to be used in Naval Dockyards.

Discussion of the Processes

Sprayed asbestos insulation

The most hazardous process involving asbestos in the Dockyard has been the spraying of environmental insulation. This was made clear by figures contained in the report of the Advisor on Applied Hygiene to the Medical Director-General in 1951.

In that report gravimetric samples showed that the dust produced during spraying of mess decks was between 155 and 640 mgm/m^3.

Particle counts showed 173-322 asbestos fibres/c.c. (fibre length 2-10 microns), and 3,121-5,957 particles/c.c. of other dusts (0.5-5 microns). Obviously this was an extremely dangerous job even though little of the dust was asbestos.

Spraying diminished in Naval Dockyards in the late 1950's and stopped in 1963, but the removal of the material has been a continuous problem. It must be remembered that between 1947 and 1967 only the men actually involved in these processes were given any form of respiratory protection, and there was no real attempt to isolate the work.

The need to remove over 750,000 sq. ft. of sprayed crocidolite asbestos from an aircraft carrier in 1967 presented an opportunity to study this process. The preventive measures employed in this operation have been described earlier in this report.

Samples were taken in the upper aircraft hangar, which was the largest area covered with sprayed asbestos, and in many of the smaller compartments, mess decks, storerooms, cabins, bathrooms and passageways leading from them.

The insulating layer, 2"-4" thick, was held in place by expanded metal clipped to the deck, and then covered with asbestos cement. To remove this layer the cement surface was broken with a small, curved crow-bar, the metal and most of the asbestos ripped away and the clips hammered off with a hammer and cold chisel. The remaining asbestos was then brushed off with wire brushes. The debris was bagged and taken out, the residue was swept up and finally the space was cleaned with industrial vacuum cleaners and damp cloths.

Table 3.A shows the dust concentrations in various compartments. The samples were taken over the working shifts, except for the fibre counts where it was discovered that the samples were far too dense to count if the sampler was run for the whole shift. The fibre counts represent samples taken over 1-2 hours of the working shift at the same positions as the gravimetric samplers.

It can be seen that there was a wide range of dust concentrations but that sweeping and bagging produced more fine dust and higher fibre counts than the actual stripping procedure. Some "snap" samples taken with the Draeger pump in the breathing zones of men bagging debris showed concentrations of between 1,000-2,000 fibres/c.c., but they are not included in this

data.

The samples taken in passageways adjacent to stripping areas show how difficult it is to contain the dust even when considerable efforts are made to do so. Men were working in these locations without respiratory protection, and it is easy to see how these so called "neighbourhood" workers could have had considerable asbestos exposure in the past when no attempts were made to contain the dust. The same pattern can be seen from the fibre counts in Table 3.B where a long running sampler was set up at one stripping area, another at the top of the ladder from this deck, and another at the top of a ladder to the deck above.

The removal of debris in sealed paper bags obviously made a difference to the overall level of dust concentration throughout the ship, but we do not have comparable figures for the old method using jute sacks which were pulled up the steps, emptied into a barge and re-used. The concentrations about the stack awaiting disposal in the open air were negligible.

Special cleansing, changing and eating facilities were made available on the ship for these workmen and Table 3.B shows that although they passed through a shower wearing their impervious overalls this did not remove all the asbestos. A man was employed to clean the overalls with a damp cloth, and this precaution seemed to be satisfactory as shown by the counts in the eating and resting areas.

<u>Summary</u>

Dust sampling has shown that spraying crocidolite asbestos was extremely dusty. The removal of the material also produced very high dust concentrations in other parts of the ship than at the site of work. The overall dust concentrations during these procedures in the past, when no real attempt was made to contain the dust, would almost certainly have been higher.

Machinery and Pipe Lagging

Removal or Stripping of Pipe Lagging

No previous figures are available for dust concentrations during stripping of old machinery insulation in the Dockyard. For the present study long period samples were taken in the machinery spaces of aircraft carriers, cruisers, and frigates, in which amosite and/or calcium silicate pipe sections covered with asbestos cloth and asbestos cement were being removed.

The gravimetric samplers were placed in a convenient place, at head height where possible, and fibre counts were taken either alongside the gravimetric samplers or at other parts of the compartments to give an idea of general concentrations. Samples were also taken with Hunt personal samplers as near as possible to the men's faces to give "breathing zone" samples.

Table 3.C shows the concentrations found in typical sites. It can be seen that all the values are higher in boiler rooms than in engine rooms. This is due to the differences between the two compartments. The boiler rooms have more insulation in comparison with the available space than do the engine rooms. Also, work progresses on at least two levels in boiler rooms so that a lot of debris falls perhaps 6-14 feet to the deck giving rise to more dust in the general atmosphere. This is also the most likely explanation for the fact that, in boiler rooms, the mean value for general atmosphere fibre concentrations is higher than the breathing zone values. The two types of concentrations are about the same in the engine rooms.

The very high counts in the brick stowage space are the result of removing a large area of insulation in a very small, enclosed space. Similar conditions are met with in funnel uptakes, behind and between boilers, in steam accumulator compartments and other small spaces where a lot of pipe work has to be insulated.

To obtain some idea of the wide fluctuations in dust concentration during the removal of pipe lagging in a boiler room, frequent samples were taken at fixed points daily for a week. The values are given in Table 3.D and the mean values from the sampling points are shown graphically in figure 3.1. The sampling points, designed to give estimates of general dust concentrations are shown in figure 3.2. For this study only fibre counts were made.

The apparently short working periods are the result of the time taken for the men to assemble their equipment and to reach the ship, the requirement for them to carry the debris out of the compartment in sealed bags at the end of each shift and the time allowed at the end of each shift for them to shower and change.

It can be seen that the concentrations fall quickly when active stripping stops, and rise rapidly again when the work restarts after the break.

It was very difficult to calculate a time weighted average concentration because of the continuously altering nature of the work and the fact that different men might be withdrawn from work in this compartment to other jobs from day to day. This is not like the conditions in an asbestos textile factory where an employee is exposed to roughly constant dust concentrations because he performs the same process throughout the workin period.

FIGURE 5.1

Asbestos fibre concentrations (fibres/c.c.) during the records of fibre loading (See Table 5.1).

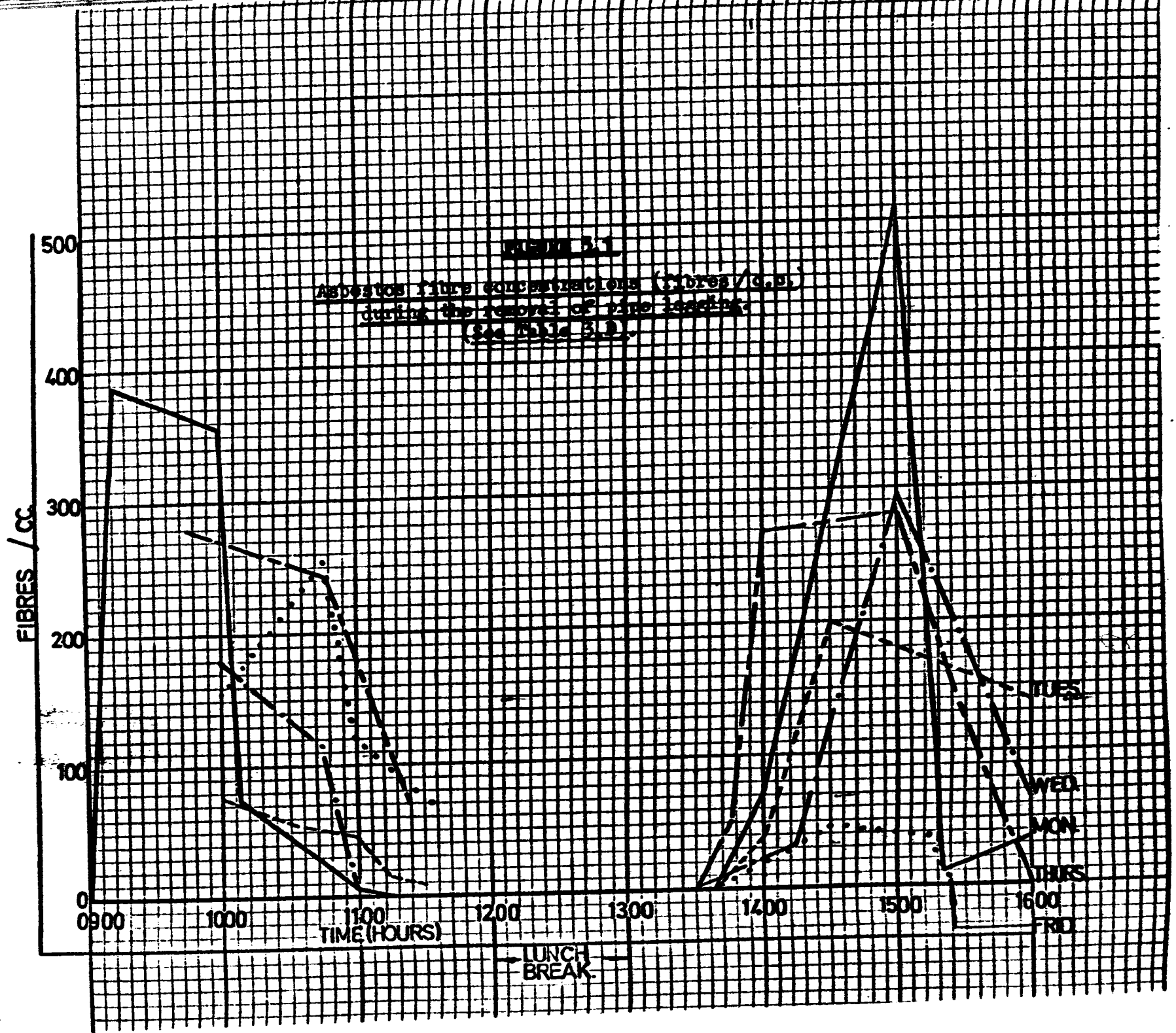
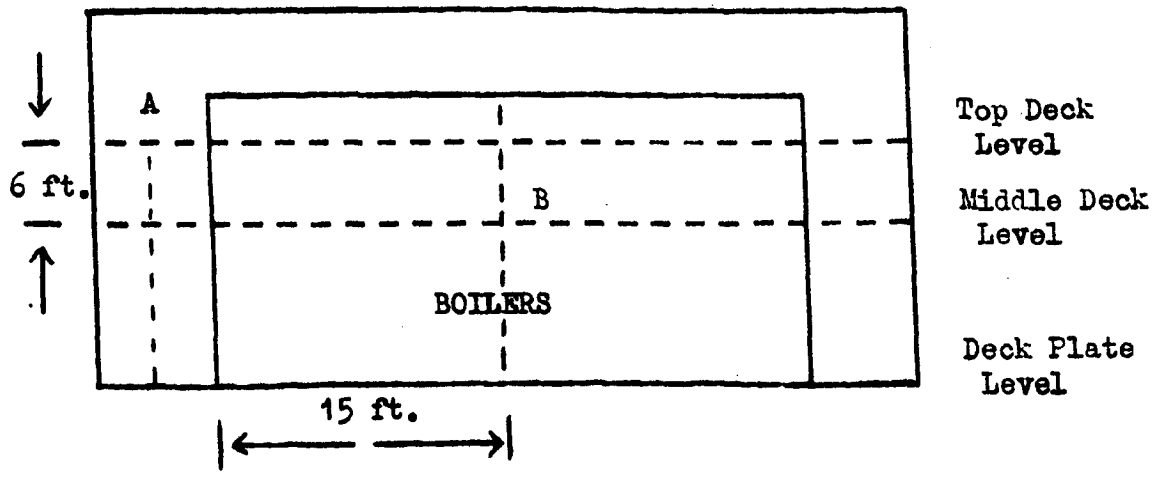


FIGURE 3.2

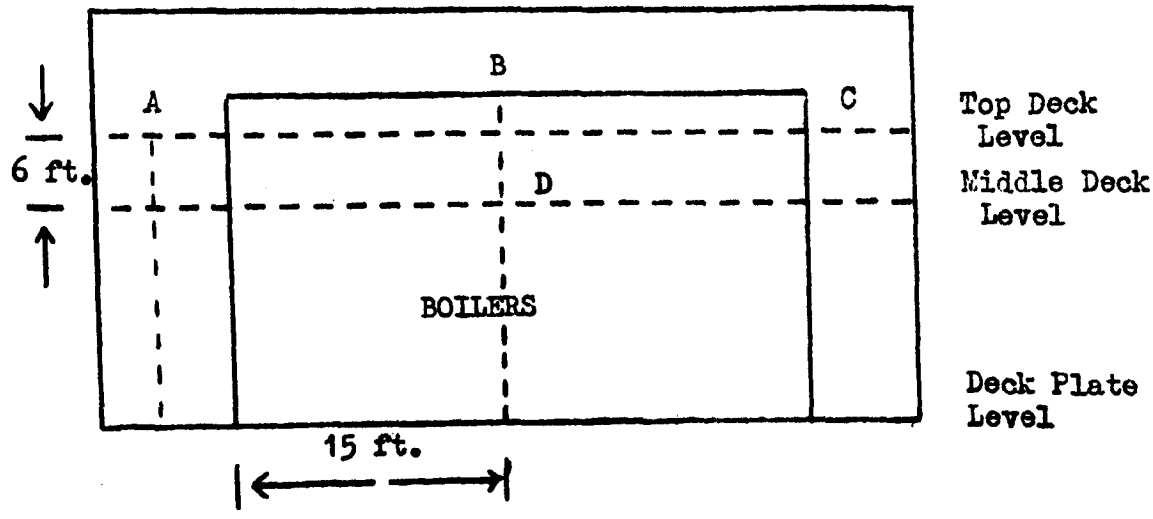
Stripping of Amosite from Boilers, Machinery and Pipes

Sampling Positions:-

MONDAY-TUESDAY



WEDNESDAY-FRIDAY



However, the average dust concentration for the 106 samples taken during the week was 121.2 fibres/c.c. and the average number of hours worked in the compartment was 24 hours so that the average time weighted concentration for the 40 hour week was $\frac{121.2 \times 24}{40}$ = 72.8 fibres/c.c. The problem of calculating time weighted concentrations will be discussed later in this report.

Application of Pipe Lagging

No values of dust concentrations were obtained for this report for the application of pure amosite sections as these are no longer recommended for use in ships of the Royal Navy. This was part of the attempt to reduce the amount of asbestos used in insulating materials, as these sections contained over 90% amosite fibre.

The materials used in the pipe lagging operations investigated were calcium silicate sections containing up to 15% amosite asbestos, asbestos rope, asbestos cloth, asbestos "plastic mix", and asbestos cement. The dust concentrations found are given in Table 3.E.

The gravimetric results show again that more dust is produced in the boiler rooms than in engine rooms and in the small steam accumulator room. More insulating materials are used, and the dropping of broken pieces of sectional material gives rise to the higher dust levels.

This is also demonstrated by the fibre counts; the general disturbance of material in the boiler rooms brings the mean general atmosphere counts very near the breathing zone concentrations. In the engine rooms the general disturbance is less than in boiler rooms and the dust levels are higher in the immediate vicinity of the worker.

Miscellaneous Processes associated with Machinery Insulation

Tables 3.F and G give the values of samples taken during various processes associated with machinery insulation.

The manipulation and sawing of calcium silicate sections gives rise to surprisingly high fibre concentrations; it was hoped that these materials would not produce high fibre counts because of the relatively small amount of asbestos in them.

Fitting amosite rope produces extremely high fibre concentrations as does the handling and mixing of plastic asbestos mixture in a bucket. The latter process should now have ceased, but it is very difficult to change long standing working habits. Another dusty job is the "blowing down" of debris with an air hose as part of the cleaning of a compartment. This is an effective way of dislodging debris from the multiple of ledges, pipes, gratings and other surfaces, and it is unlikely that a suitable alternative method will be found. Similarly, although vacuum cleaners are being increasingly used, the sweeping and bagging processes will probably be retained and will continue to produce very high dust levels.

Few samples were taken during the ripping of asbestos cloth, but they suggest that the treatment of the cloth with a dust suppressant by the manufacturers has been very effective in reducing the amount of dust produced in this process. The figures relating to the tearing of contaminated cloth shows that cloth left lying about picks up many fibres from the settling debris and these are disturbed again by the tearing procedure.

In Table 3.H, the gravimetric samples taken during the removal of friable asbestos acoustic panels include very high values, but most of

This dust was not of respirable size. The reason for the high general atmosphere fibre counts, also shown in Table 3.H, is that they were taken some five feet from the deck while the breathing zone samples were taken up at the deckhead where the men were working on staging and throwing the debris to the deck.

Table 3.I shows some fibre counts in minor processes using asbestos. The high counts found during the use of asbestos cloth to protect equipment from welding spatter were due to the habit of brushing and chipping the slag off the cloth so that it could be used again.

The asbestos mattress shop was removed to a new purpose built shop during the survey but the results shown in Table 3.J indicate that there is very little to choose between the old and the new buildings. In each case there are some high fibre counts and these were shown to be due to the sweeping up of amosite fibre around the filling cabinets.

In asbestos stores the dust concentrations were very low except for the brief periods in which stacking or removing pipe sections took place. High counts were also found when debris was swept up into a bucket, and varied between 36-128 fibres per c.c. These data do not appear in the Tables.

Other data not shown in these Tables were the dust concentrations found during work on a submarine. Dust suppressed asbestos cloth was extensively used for maintaining heat in a steel bulkhead while a watertight door was being welded into place. It was feared that disturbance of the cloth might liberate asbestos fibre, or that the

exhaust from the compressed air caulking tools would blow clouds of fine asbestos dust into the general atmosphere. There was also concern that the prolonged heating of the cloth (100°C for 58 hours) would reduce the efficiency of the dust suppressant and allow the cloth to shed asbestos fibre while being removed and placed into paper sacks for removal.

Six samples of between 300-900 c.c. were taken by the Draeger pump close to the cloth while it was being cut and fitted into position around the heating elements. All counts were below 1 fibre per c.c. Personal samplers were worn by the two men doing this work, with similar results.

Five samples were taken close to the cloth with the Draeger pump while caulkers were working on the weld, and six long period samples were also taken. All of these counts were below 1 fibre per c.c. By this time the cloth had been heated to 100°C for 58 hours. Long running samplers were worn by the men removing the cloth from the heating coils. The values of these counts and a Draeger sample close to the cloth, were below 1 fibre per c.c. Two samples were taken with the Draeger pump when the men were putting the used cloth into the container for disposal. One count was below 1 fibre per c.c. and the other 2.02 f/c.c.

The above results show lower dust concentrations associated with the pre-heating welding process than do the results of an earlier series of samples taken on an aircraft carrier. (Table 3.I). The earlier samples were taken when the men were using asbestos cloth not treated with a dust suppressant. The cloth was also subjected to constant disturbance by being walked on and moved about to give access to the work.

Table 3.K shows the results of samples taken in a boiler room which was partly insulated. The laggers were applying the insulation at night and other tradesmen worked in the compartment during the day. The latter were thought to be safe and were not issued with dust respirators. High local concentrations were found when men were clambering over partly insulated pipes. Lower values were discovered during the cleaning of debris with a vacuum cleaner, but these were general atmosphere samples taken with the Draeger pump, and were for a short period only. The long period 'personal' samples taken in the breathing zones of fitters working on a partly insulated pipe are quite low and the long running samples of the general environment are extremely low.

Comparison of Mass and Fibre Concentration

The technique of membrane filter fibre counting takes time and the services of an experienced technician if reliable results are required. It is easier, quicker, and more accurate to weigh the dust.

It was thought to be unlikely that we could find a relationship that would allow the prediction of fibre concentration from the weight of total or respirable dust because of the different amounts of asbestos contained in the many products used in the various processes.

To see if this assumption was correct long running membrane filter samplers were used alongside gravimetric samplers during the removal of sprayed asbestos, the removal of pipe lagging and the application of pipe insulation. The values of fibre counts, 'total' dust weights and 'respirable' dust weights are given in Tables 3.L, M and N.

Plots of fibre counts against 'total' dust, and against 'respirable' dust suggested that there was not a close relationship between them. I am indebted to Mr. P.D. Oldham for analysing this data, and his calculations confirm that it is not possible to derive any one of the indices from another.

However, the mean concentrations in each compartment are roughly in the same rank order and show a fair degree of correlation (Fig. 3.7).

If it is assumed that respirable mass and fibre counts are in the same proportion to each other it might be possible to assess the proportional hazard of times spent in each type of compartment and process. The best fitting constant of proportionality between fibre count and respirable mass is 14.7:1, and on this basis the relative dust concentrations are:-

Location		Relative Concentration
Application:	Engine rooms	1
	Boiler rooms	2
	Accumulator compartment	1
Stripping:	Sprayed limpet asbestos	11
	Brick stowage space	57
	Boiler rooms	7
	Engine rooms	3

It must be emphasized that the reliability of these weights is probably very small, but they do perhaps fit fairly well with the impression of relative dustiness we have gained during the observations of the various processes.

DISCUSSION

These results give some idea of the conditions that are likely to have occurred in Naval ships refitting during the last 20 years. It is impossible to give an accurate assessment of past dust concentrations, but it is probable that conditions in the past were worse than those discovered during this survey.

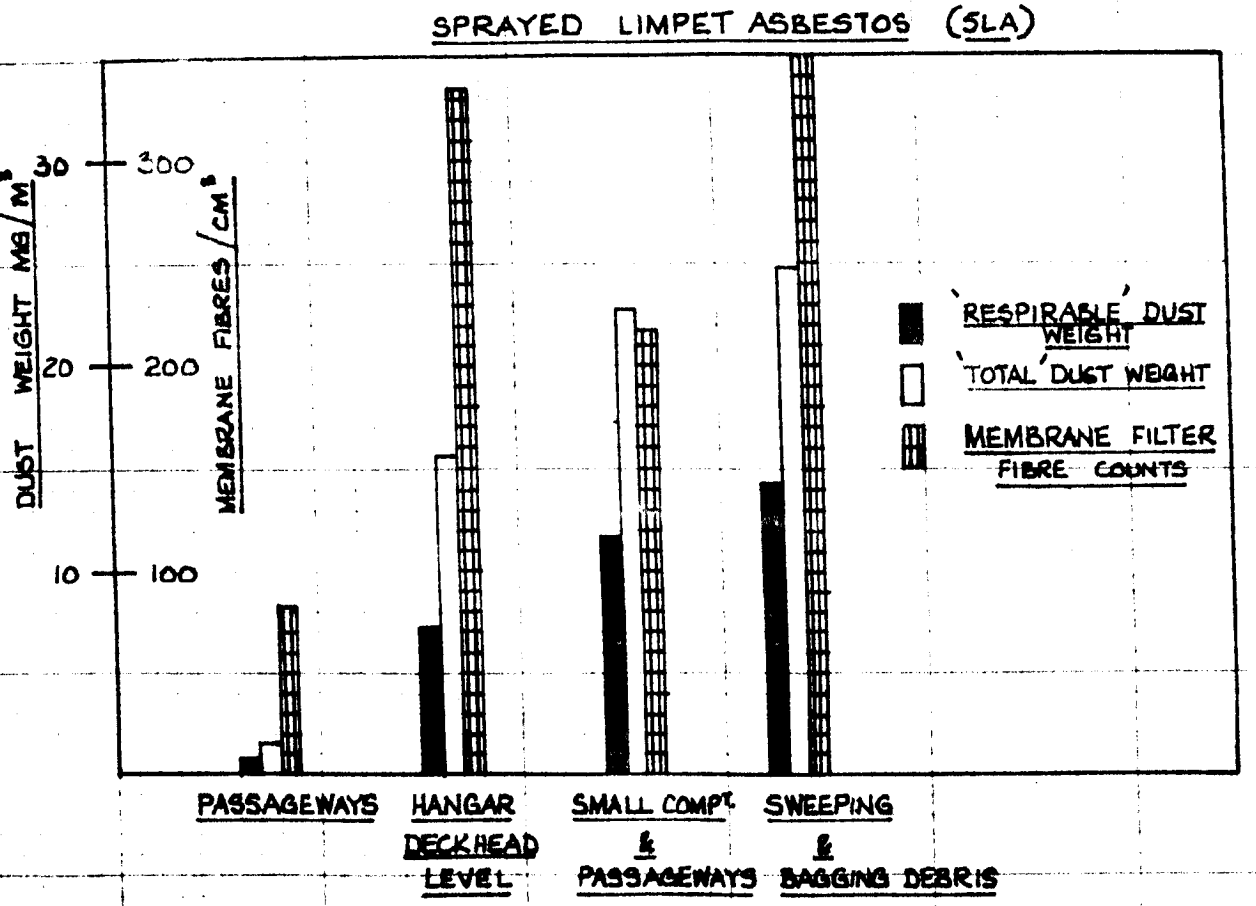
The gravimetric samplers give a measure of overall dustiness and the membrane filter counts show that the dust contains high concentrations of asbestos fibre, especially during the stripping procedures. (Fig. 3.3). The removal of sprayed crocidolite asbestos must be considered a most hazardous process. The extent of the hazard can be realised when the values for the fibre counts are compared with the provisional threshold limit of 0.2 f/c.c. proposed by the Factory Inspectorate. The preventive measures taken to protect the men doing this work have been described. The dust sampling results help to show that it is very difficult to confine the dust to the actual stripping area, and explains how many men not thought to have been at risk in the past may have had considerable exposure to blue asbestos fibre. It is encouraging to know that this process is becoming increasingly rare as few Royal Naval ships now contain crocidolite asbestos.

The dust concentrations during the removal of machinery insulation are also very high, and this again helps to emphasize the considerable asbestos exposure that the so called "neighbourhood" workers have been intermittently subjected to for many years. Sweeping, "blowing down", and bagging debris cause very high dust concentrations and until recently the men performing these procedures would not have had respiratory protection.

The dust levels during the application of pipe and machinery insulation are understandably lower (Fig. 3.4), but mixing asbestos cement, handling

FIGURE 3.3

MEAN DUST CONCENTRATIONS DURING REMOVAL OF ASBESTOS INSULATING MATERIALS



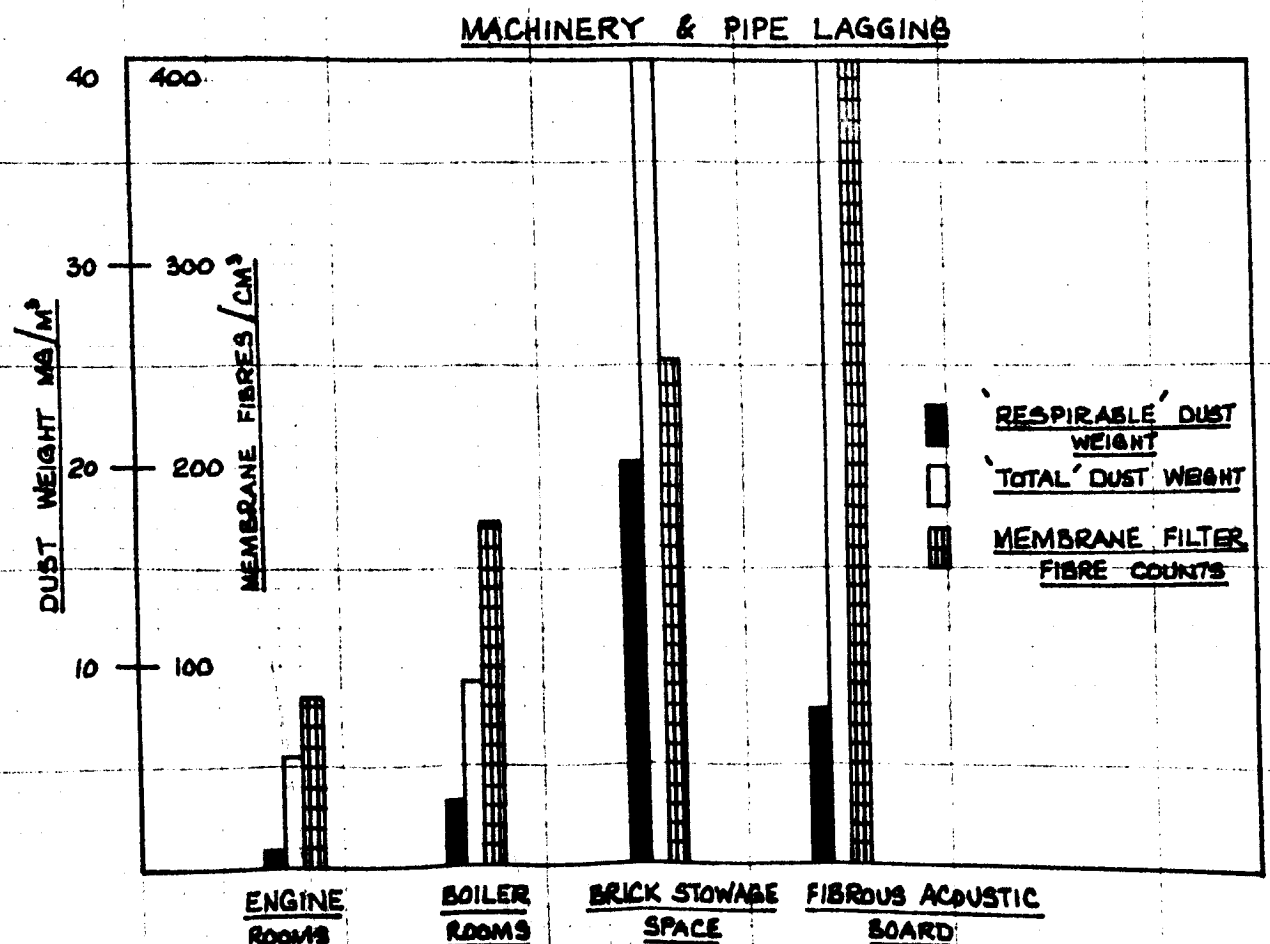
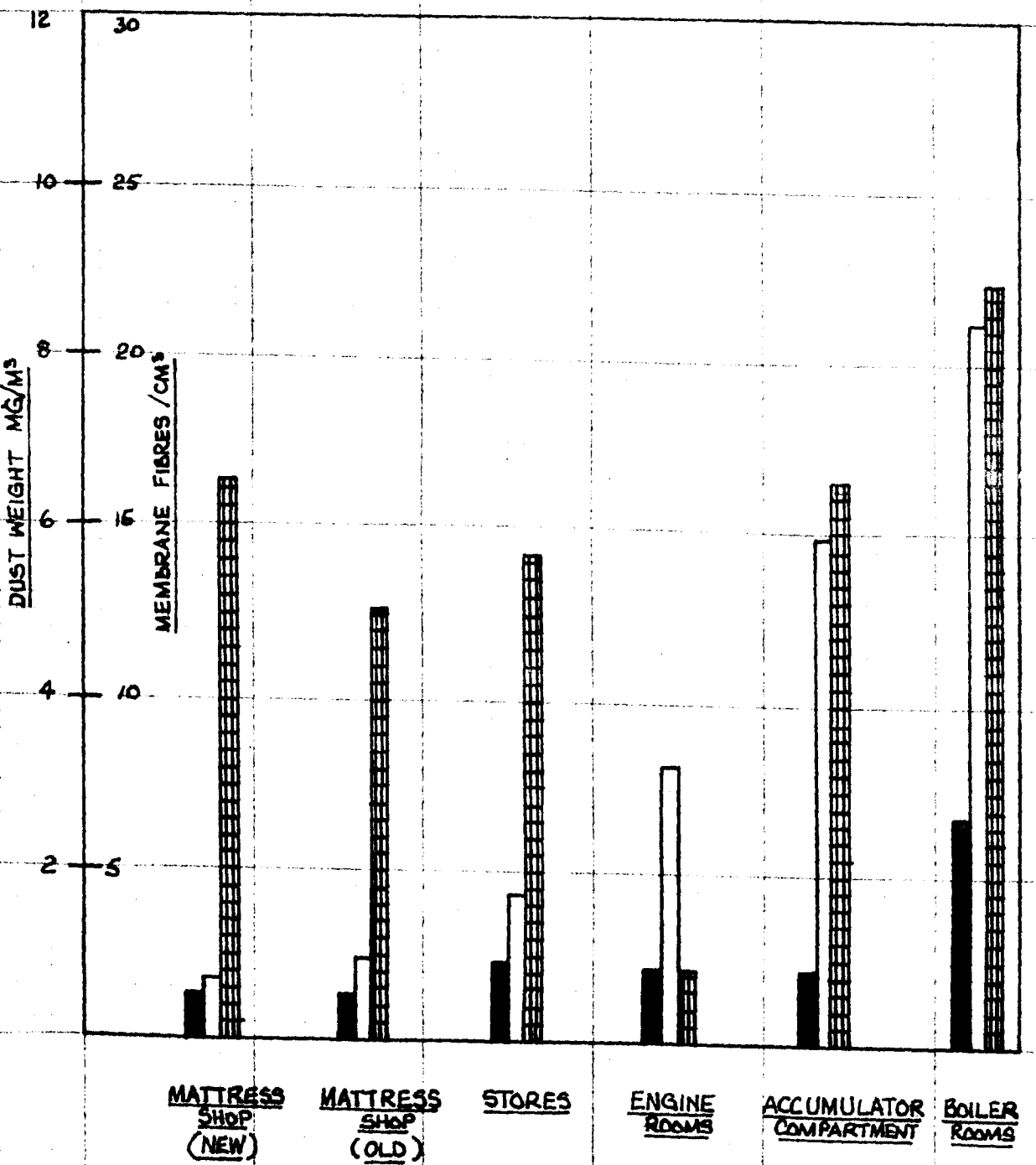


FIGURE 3.4

MEAN DUST CONCENTRATIONS IN MATTRESS SHOPS, STORES AND DURING APPLICATION OF PIPE LAGGING



and sawing pipe sections are dusty procedures which help to raise the general atmosphere dust concentrations (Fig. 3.5).

The sampling results have shown that the use of asbestos cloth by welders to protect equipment from hot metal slag is only hazardous when the cloth is shaken or brushed to remove the slag in order to use the cloth again (Fig. 3.6). The cloth treated with a dust suppressant and discarded without undue disturbance produces very low concentration of asbestos fibre, and this is now recommended in the code of practice for welders and burners. Similarly, sampling has shown that the use of dust suppressed asbestos cloth for the pre-heating welding techniques does not cause high dust concentrations. Instructions have been given to minimize the disturbance of the cloth which is discarded after use.

Guillotining perforated asbestos board in the workshop instead of sawing it on board the ship has been shown to help to reduce the amount of asbestos fibre liberated into the atmosphere.

The dust levels have helped to confirm the assessment of the degree of risk associated with the various processes. These are set out in Figures 3.8,9 and 10 together with the warning notices displayed and the protective equipment provided for the workers. These regulations are now enforced in all Naval Dockyards.

It is very difficult to attempt to produce time weighted averages of asbestos exposure for Dockyard insulation workers. It is virtually impossible to do so for the "neighbourhood" workers. It is therefore extremely difficult to relate these results to the provisional TLV of 2 f/c.c. proposed by HM Factory Inspectorate. On a time weighted basis over 3 months it is possible that all the "neighbourhood" workers, and the

FIGURE 3.5

MEAN DUST CONCENTRATIONS OF MISCELLANEOUS PROCESSES DURING APPLICATION OF PIPE LAGGING

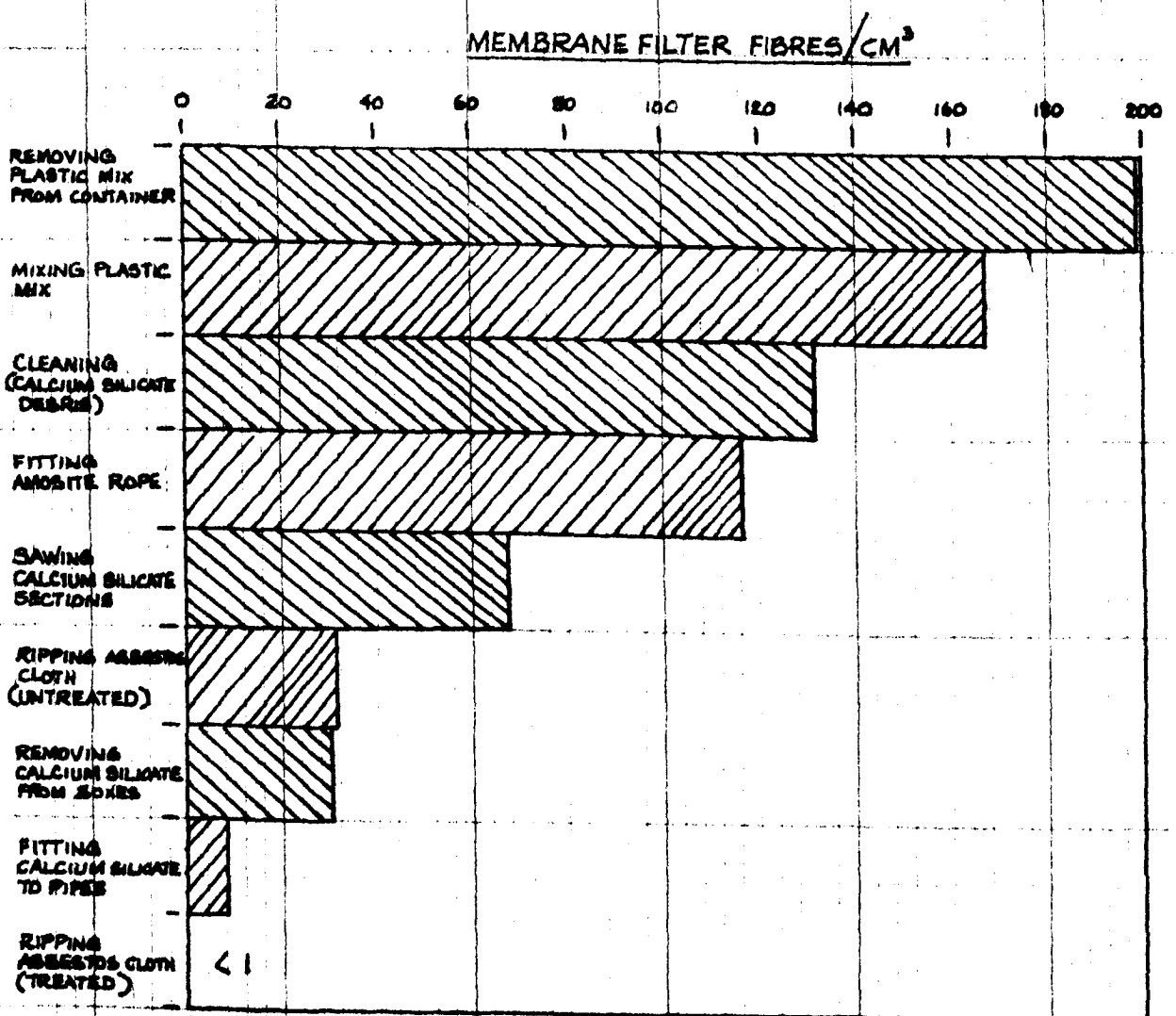
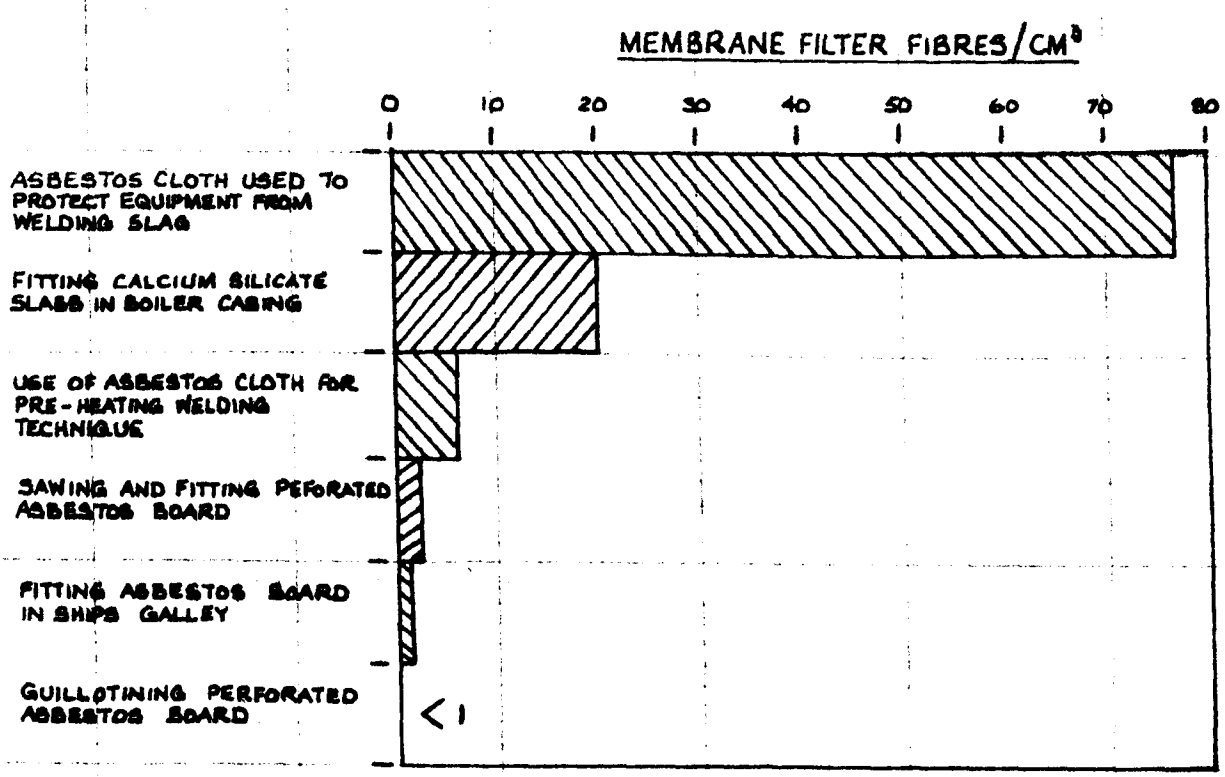


FIGURE 3.6

MEAN DUST CONCENTRATIONS IN OTHER MISCELLANEOUS PROCESSES INVOLVING ASBESTOS



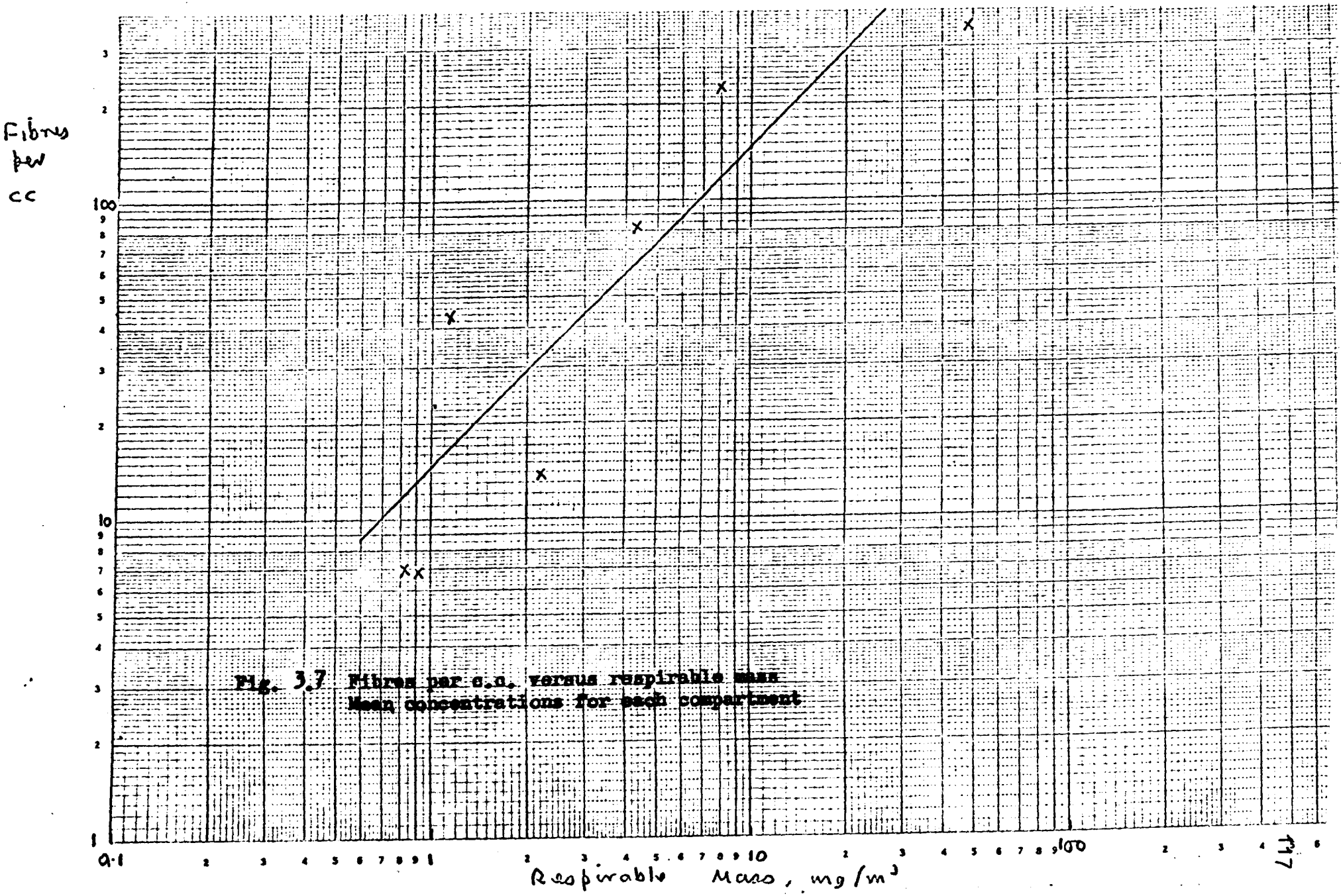


Fig. 3.7 Fibres per c.c. versus respirable mass
Mean concentrations for each compartment

FIGURE 3.8

PROTECTIVE MEASURES FOR MOST HAZARDOUS PROCESSES

PROCESS	REMOVAL OF SPRAYED CROCIDOLITE ASBESTOS	REMOVAL OF LARGE AMOUNTS OF PIPE LAGGING
NOTICES	SPRAYED ASBESTOS REMOVAL NO ENTRY	ASBESTOS DE-LAGGING NO ENTRY
REGISTERED ASBESTOS WORKERS	IMPERVIOUS SUIT AIR FED RESPIRATOR SHOWERS	IMPERVIOUS SUIT AIR FED HOOD OR POSITIVE PRESSURE POWER RESPIRATOR SHOWERS
NEIGHBOURHOOD WORKERS	NOT ALLOWED ENTRY IF ENTRY IS VITAL, PROTECTION AS FOR REGISTERED WORKER	
SUPERVISORS	NYLON/PLASTIC SUIT. FULL FACE PIECE RESPIRATOR WITH HOOD OR POSITIVE PRESSURE POWER RESPIRATOR.	
VISITORS	VISITS TO BE LESS THAN 30 MINUTES. IF LONGER PROTECTION AS FOR REGISTERED WORKER.	

FIGURE 3.9

PROTECTIVE MEASURES FOR HAZARDOUS PROCESSES

PROCESS	APPLICATION OF LARGE AMOUNTS OF PIPE LAGGING	APPLICATION OR REMOVAL OF SMALL AMOUNTS OF PIPE LAGGING
NOTICES DISPLAYED	ASBESTOS WORK IN PROGRESS RESPIRATORS TO BE WORN	ASBESTOS WORK IN PROGRESS
REGISTERED ASBESTOS WORKERS	NYLON SUIT. APPROVED DUST RESPIRATOR SHOWERS	
NEIGHBOURHOOD WORKERS	PREFERABLY NOT ADMITTED IF ENTRY REQUIRED PROTECTION AS FOR REGISTERED WORKER	NO RESTRICTION ON ENTRY RESPIRATORS AVAILABLE
SUPERVISORS VISITORS	NO RESTRICTION ON ENTRY NYLON SUITS AND RESPIRATORS AVAILABLE	

FIGURE 3.10

PROTECTIVE MEASURES FOR LEAST HAZARDOUS PROCESSES

PROCESS	ASBESTOS MATTRESS SHOP	ASBESTOS STORES	OTHER MINOR ASBESTOS PROCESSES
NOTICES DISPLAYED	ASBESTOS SHOP NO UNAUTHORIZED ENTRY	ASBESTOS STORE NO UNAUTHORIZED ENTRY	ASBESTOS WORK IN PROGRESS
REGISTERED ASBESTOS WORKERS	NYLON SUIT APPROVED DUST RESPIRATOR SHOWERS		
NEIGHBOURHOOD WORKERS	RESPIRATORS AVAILABLE		
SUPERVISORS VISITORS	RESPIRATORS AVAILABLE		

majority of laggers might have a calculated time weighted average exposure of less than 2 f/c.c., but during that time they might have been exposed to very high concentrations of dust for short periods (Fig. 3.1). If another 3 monthly period was taken for study it is quite possible that most of the laggers, and in the past many "neighbourhood" workers, might have been exposed to the same range of concentrations given in Table D and Figure 1 for the whole of the time, resulting in a time weighted average similar to that previously calculated for one week from these data, i.e. 72.8 f/c.c.

It seems reasonable to surmise that over the last 20 years or so, with little or no attempt to control the dust hazard, many men have been exposed for long periods to average concentrations well above the proposed TLV of 2 f/c.c.

The results obtained in the studies of other shipyard insulation processes, which have previously been reviewed in this report, are similar to those we have found in this Dockyard. The overall dust levels described by Fleischer et al were very high. He does point out that there were varying amounts of asbestos in the materials and the results show that few fibres were found by the Konimeter in relation to the total counts. Our experience of this instrument is that it underestimates the fibrous particles present in our dust clouds when compared with membrane filter counts. While agreeing with Fleischer and his colleagues that total dust counts of below 5 mppcf probably indicate good dust control, his results show that only 4 out of the 25 mean values for total dust were below this level. Five values were between 5-10 mppcf; twelve between 10-50 mppcf; and five over 50 mppcf. Murphy and Ferris in their 1966 survey found no total dust values lower than 5 mppcf using the Konimeter. All these results relate to processes associated with the application of new insulating

materials. The dust concentrations during removal of these materials must have been higher.

It is probably impossible to reduce the dust concentrations during insulating work in ships to below the level of 2 f/c.c., but the practical methods of protecting the workmen and isolating the processes which have been developed during this study should help to reduce the number of men affected by the diseases associated with asbestos.

Because of the continued requirement to remove existing insulating material for the next 10 years or so, each Naval Dockyard is now setting up dust sampling units in order to monitor the work environment so that proper supervision of the protective measures can be enforced. The technique that has been recommended for this purpose is the standard membrane filter fibre count method. Gravimetric methods have been shown to give a picture of overall dustiness, but not to give an accurate estimate of asbestos exposure because of the varied materials used in Dockyard insulating processes.

Probably the first Naval frigate in the world to have no asbestos insulation for its machinery or pipe work is at present being built in Devonport Dockyard. If the substitute materials (asbestos free magnesia and asbestos free calcium silicate sections, glass fibre cloth and asbestos free hard setting cement) prove to be robust enough to be acceptable to the engineers, then a very big step forward will have been taken towards eliminating the problems associated with asbestos in the Dockyards.

Photography of Dust Clouds

The photography of dust produced in the processes described in this report is difficult, and a special technique is required. Such a technique has been devised by Mr. W.B. Lawrie, HM Engineering Inspector of Factories (Lawrie 1951, Lawrie et al 1953). Figures 3.11-3.14 are examples of his work. The photographs clearly show the amount of dust produced, and it is only by using the special lighting technique that the dust can be made obvious.

No further explanation is necessary as these photographs explain themselves. Dust sample values are expressed in fibres per c.c. using the membrane filter technique.

I am grateful to Mr. Lawrie for his permission to reproduce these photographs from his 35 mm cine film of dust produced in insulating processes.

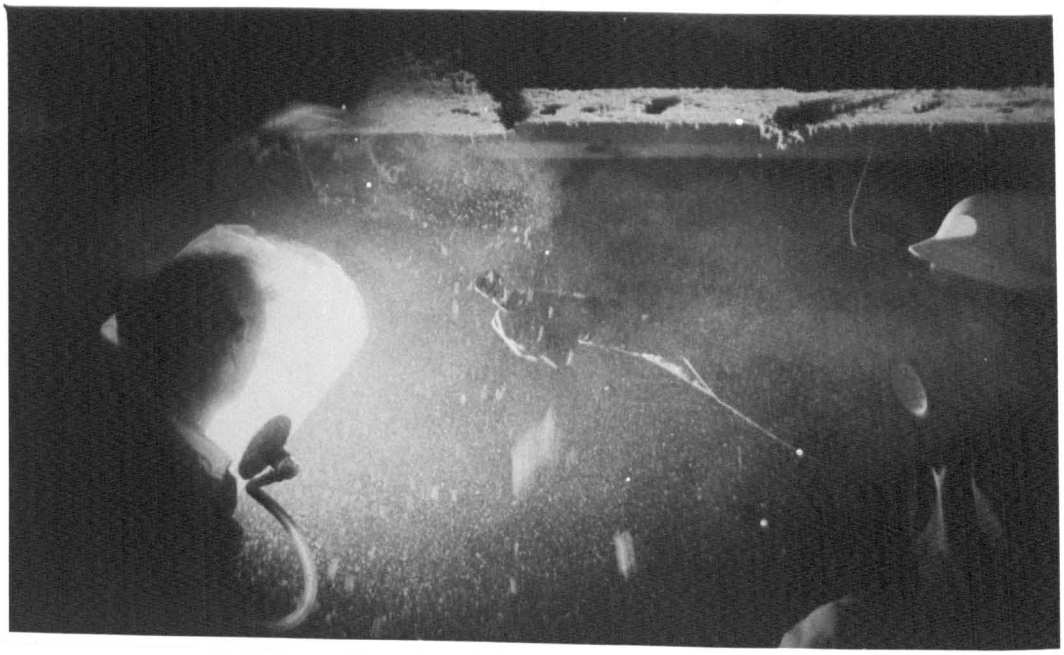


Fig. 3.11 Removing sprayed crocidolite asbestos. Workman wears airfed hood. Dust sample being taken. Result showed 1.012 asbestos fibres per cc.

Fig. 3.12 Dust billowing out of compartment in which sprayed asbestos was being removed.

Fig. 3.13 Bagging sprayed crocidolite asbestos debris. Dust sample showed 3.815 fibres per cc.

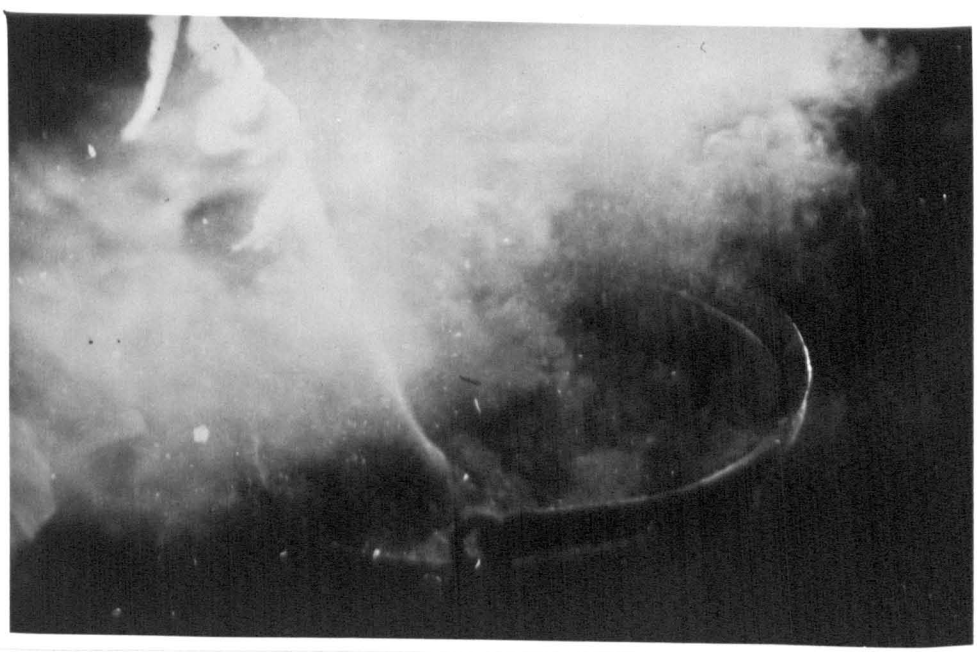


Fig. 3.14 Dense cloud of asbestos fibre and cement as this mixture was mixed in a bucket.

TABLE 3.A

DUST CONCENTRATIONS WHILE STRIPPING SPRAYED CROCIDOLITE ASBESTOS

Location	No. of Samples	M.R.E. Samplers				Fibres/c.c.	
		Total Dust Wt. mg/M^3		Respirable Dust Wt. mg/M^3			
		Mean Value	Range	Mean Value	Range	Mean Value	Range
Hangar (at deck head level)	5	15.55	6.90- 27.7	7.04	4.30-13.20	334.0	117-484
Small compart-ments and passage ways	17	22.91	9.72-103.7	11.96	2.26-33.70	x 219.9	x 35-384
Sweeping and bagging debris	2	24.80	22.10- 27.4	14.20	8.20-20.20	353.0	213-493
Passage ways adjacent to stripping areas	4	1.47	1.02- 2.0	0.82	0.46- 1.42	82.6	43-177
Passage way (Removal of sealed bags of debris)	1	1.20	-	0.48	-	23.0	-
Stacking bags of debris on upper deck	1	1.06	-	0.10	-	0.1	-
Hangar (outside stripping area)	4	-	-	-	-	56.5	19-131

x Only 15 samples evaluated - Fibre distribution too dense to count in two samples.

TABLE 3.B

FIBRE COUNTS - STRIPPING SPRAYED CROCIDOLITE ASBESTOS

Location	Sampler	Fibres/c.c.
No. 7 Deck Stripping Area	Hunt	311.00
Hatchway No. 6 Deck Leading from No. 7 Deck	Hunt	109.00
Hatchway No. 5 Deck Leading from No. 6 Deck	Hunt	30.00
Passageway Leading to Shower	Hunt	25.00
Entrance to Shower	Draeger	10.00
Removing Suits after Showering	Hunt	16.00
Workmans Dining Room	Draeger	0.10
Mens Mess Area	Hunt	0.05

TABLE 3.C

DUST CONCENTRATIONS DURING REMOVAL OF PIPE LAGGING

GRAVIMETRIC VALUES mgm/m^3

Location	No. of Samples	Total dust mgm/m^3		Respirable dust mgm/m^3	
		Mean	Range	Mean	Range
Boiler Rooms	44	9.20	1.94-46.69	3.19	0.61-16.21
Engine Rooms	12	5.33	0.58-21.00	0.96	0.38- 2.71
Brick Stowage Space	4	41.54	35.50-56.10	20.05	14.30-25.60

MEMBRANE FILTER COUNTS FIBRES/c.c.

Location	General Atmosphere Samples f/c.c.			Breathing Zone Samples f/c.c.		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Boiler Rooms	153	171.0	0.04-1062.0	20	97.1	24.7- 22.0
Engine Rooms	45	88.3	0.16-3021.0	25	90.6	2.0-490.0
Brick Stowage Space	13	256.8	9.10- 591.9	-	-	-

TABLE 3.D

DAILY TABULATION

MONDAY				TUESDAY				WEDNESDAY				THURSDAY				FRIDAY			
Time	Sampling Position	Fibres/ c.c.	Mean Fibres/ c.c.	Time	Sampling Position	Fibres/ c.c.	Mean Fibres/ c.c.	Time	Sampling Position	Fibres/ c.c.	Mean Fibres/ c.c.	Time	Sampling Position	Fibres/ c.c.	Mean Fibres/ c.c.	Time	Sampling Position	Fibres/ c.c.	Mean Fibres/ c.c.
0900	B	10.59	10.59	1000	A	100.70	78.42	1000	A	264.30	179.40	0945	A	96.10	279.40	1000	A	120.10	154.60
					B	56.15			B	133.70			B	298.10			C	171.90	
									C	184.00			C	343.80			D	171.90	
									D	65.39			D	379.90					
0912	A	317.40	381.30	1030	A	21.67	56.60	1045	A	46.12	116.80	1015	A	298.00	265.30	1045	A	304.40	254.80
	B	445.20			B	91.54			B	98.54			C	232.70			C	186.50	
									C	169.30							D	273.50	
									D	65.39									
1000	A	446.60	352.40	1100	A	35.80	48.41	1100	A	4.50	3.90	1045	A	185.90	240.70	1100	A	190.60	122.20
	B	258.30			B	61.03			C	3.40			C	295.60			C	79.14	
																	D	106.90	
1009	A	77.05	71.50	1115	A	17.08	17.52	1330	B	1.20	1.20	1115	A	76.90	85.70	1125	A	61.76	70.70
	B	65.90			B	17.97							C	94.50			C	89.39	
																	D	61.17	
1100	A	6.74	3.70	1130	A	10.20	9.70	1415	A	32.69	32.14	1330	D	6.07	6.07	1330	B	0.337	0.337
	B	0.60			B	9.30			B	30.77									
									C	34.61									
									D	30.49									
1115	A	0.299	0.31	1340	A	0.14	0.10	1500	A	511.60	293.40	1345	A	49.04	49.04	1345	B	45.30	27.40
	B	0.321			B	0.06			C	276.40							D	9.50	
									D	92.32									
1340	A	0.040	0.081	1400	A	40.27	33.56	1600	A	53.19	66.44	1400	A	487.60	267.40	1430	A	73.40	48.18
	B	0.123			B	26.85			C	74.99			C	88.47			C	28.66	
									D	71.14			D	227.20			D	42.56	
1400	A	63.48	72.13	1430	A	244.10	234.90	1615	B	34.61	34.61	1500	A	342.90	289.20	1515	A	39.40	41.33
	B	81.79			B	225.80							C	310.90			C	49.04	
													D	213.80			D	35.56	
1500	A	390.00	509.00	1600	A	169.30	129.90					1530	B	142.60	126.60	1530	B	4.30	4.30
	B	628.00			B	90.50							C	110.60					
1520	A	17.30	12.60	1630	A	17.69	9.09					1600	B	6.07	6.07				
	B	7.93			B	0.50													
1600	A	44.14	38.80																
	B	33.57																	

TABLE 3.E

DUST CONCENTRATIONS DURING THE APPLICATION OF PIPE LAGGING

GRAVIMETRIC VALUES mgm/m^3

Location	No. of Samples	Total Dust mgm/m^3		Respirable Dust mgm/m^3	
		Mean	Range	Mean	Range
Boiler Rooms	18	8.58	0.31-45.75	2.73	0.15-12.5
Engine Rooms	18	3.24	0.30-15.45	0.86	0.14- 2.68
Accumulator Room	13	5.30	1.19-26.62	0.87	0.52- 2.32

MEMBRANE FILTER COUNTS FIBRES/c.c.

Location	General Atmosphere Samples f/c.c.			Breathing Zone Samples f/c.c.		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Boiler Rooms	17	22.4	1.0-61.0	14	16.75	0.1 -68.0
Engine Rooms	28	2.1	0.1-14.0	16	7.30	0.04-40.0
Accumulator Room	5	16.5	2.5-45.7	17	9.56	0.99-47.1

TABLE 3.F

DUST CONCENTRATIONS IN MISCELLANEOUS PROCESSES ASSOCIATED WITH PIPE LAGGING

FIBRE COUNTS FIBRES/c.c.

Process	General Atmosphere Samples f/c.c.			Breathing Zone Samples f/c.c.		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Sawing calcium silicate sections	7	67.9	0.7 -158.0	11	54.6	6.9-152.0
Removing calcium silicate sections from box	7	30.9	2.2 - 77.6	10	51.9	16.0-136.0
Fitting calcium silicate sections to pipes	9	4.1	0 - 23.4	20	43.1	0.9-123.6
Cleaning calcium silicate debris	9	133.7	32.0 -372.0	7	154.7	90.0-277.0
Fitting amosite rope	4	117.9	1.05-279.8	13	111.7	5.0-340.0
Removing asbestos 'plastic mix' from container	7	199.8	48.0 -328.0	13	217.5	48.0-471.2
Mixing asbestos 'plastic mix' with water in bucket	3	167.1	53.0 -377.4	12	256.0	24.0-578.9

TABLE 3.G

DUST CONCENTRATIONS IN MISCELLANEOUS PROCESSES ASSOCIATED WITH PIPE LAGGING

FIBRE COUNTS FIBRES/c.c.

Process	General Atmosphere Samples f/c.c.			Breathing Zone Samples f/c.c.		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Ripping cloth (untreated)	2	33.0	23.0- 43.0	5	6.6	0.3- 16.5
Ripping cloth (contaminated)	-	-	-	12	20.1	5.5- 42.7
Ripping cloth (treated)	12	>1	>1	12	>1	>1
Stitching cloth	-	-	-	12	3.4	0 - 10.0
Fitting cloth over lagged pipes	-	-	-	7	21.6	0.3- 43.0
"Blowing down" asbestos debris	7	489.4	140.0- 932.0	-	-	-
Sweeping and bagging amosite debris	10	563.6	76.3-1191.0	-	-	-

A

TABLE 3.H

DUST CONCENTRATIONS DURING REMOVAL OF FIBROUS ASBESTOS ACOUSTIC PANELS

GRAVIMETRIC VALUES mgm/m^3

No. of Samples	Total Dust mgm/m^3		Respirable Dust mgm/m^3	
	Mean	Range	Mean	Range
4	54.31	11.5-116.9	7.94	2.28- 13.58

MEMBRANE FILTER COUNTS FIBRES/c.c.

No. of Samples	General Atmosphere Samples		Breathing Zone Samples	
	Mean	Range	Mean	Range
6	413.5	29.3-683.2	130.97	48.1-270.9

TABLE 3.I

DUST CONCENTRATIONS IN OTHER MISCELLANEOUS PROCESSES INVOLVING ASBESTOS

FIBRE COUNTS FIBRES/c.c.

Process	No. of Samples	Mean	Range
Sawing and fitting perforated asbestos board	9	2.43	0 - 10.4
Guillotining perforated asbestos board	8	0.10	0.04- 0.5
Fitting asbestos board in ship's galley	6	1.82	0 - 11.0
Use of asbestos cloth for pre-heating welding technique	9	8.72	0 - 30.1
Asbestos cloth used to protect equipment from welding slag	18	76.60	0 -660.0
Fitting calcium silicate slabs in boiler casing	18	20.80	1.10- 73.0

TABLE 3.J

DUST CONCENTRATIONS IN ASBESTOS MATTRESS SHOPS

GRAVIMETRIC VALUES mgm/m^3

Location	Total Dust mgm/m^3			Respirable Dust mgm/m^3		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Old Shop	10	0.93	0.56-1.6	10	0.53	0.27-0.76
New Shop	16	0.65	0.17-1.2	13	0.29	0.08-0.70

FIBRE COUNTS FIBRES/c.c.

Location	General Atmosphere Counts f/c.c.			Breathing Zone Samples f/c.c.		
	No. of Samples	Mean	Range	No. of Samples	Mean	Range
Old Shop	12	12.7	0 -125.8	15	1.54	0- 7.3
New Shop	11	16.3	2.0- 83.0	25	3.65	0-17.0

TABLE 3.K

DUST CONCENTRATIONS IN A PARTLY INSULATED BOILER ROOM

MEMBRANE FILTER SAMPLES. FIBRES PER c.c.

Location	Sampler	No. of Samples	Mean f/c.c.	Range f/c.c.
In breathing zones of men working on or climbing over partly insulated pipes	Draeger	8	50.80	6.00-144.00
General samples during cleaning debris with vacuum cleaner	Draeger	2	24.00	20.00- 28.00
Breathing zone of fitter working on partly insulated pipe	Hunt (Long running)	2	3.50	2.00- 5.00
General atmosphere samples	Hunt (Long running)	4	0.86	0.23- 1.83

TABLE 3.L

SPRAYED CROCIDOLITE; ASBESTOS CEMENT. STRIPPING OF SPRAYED ASBESTOS.

Gravimetric samples mgm/m^3		Fibres per c.c.
Total dust	Respirable dust	
24.1	6.0	192
17.8	7.2	160
1.2	0.5	23
22.1	8.2	213
27.4	20.2	493
10.1	2.9	117
2.9	1.7	93
27.7	13.2	371
17.6	9.0	484
21.7	3.3	58
1.7	0.8	43
23.9	3.3	61
2.0	1.4	54
9.7	3.0	210
1.0	0.5	177
1.2	0.5	57
12.5	5.1	120
37.4	18.4	311
6.9	4.3	296
15.5	6.8	412
24.1	16.4	375
23.0	15.7	384
10.1	3.7	110
45.4	23.9	343
13.3	10.8	360
49.7	20.1	366

TABLE 3.M

AMOSITE SECTIONS AND ASBESTOS CLOTH. STRIPPING OF PIPE LAGGING.

Location	Gravimetric samples mgm/m^3		Fibres per c.c.
	Total dust	Respirable dust	
Boiler Rooms	7.60	3.21	24.7
	6.19	3.26	56.9
	13.54	3.13	68.0
	7.61	2.14	41.9
	46.69	16.21	152.2
	24.66	9.72	123.0
	4.14	1.85	66.5
	11.73	1.38	50.7
	9.80	3.61	186.4
	17.32	3.95	135.0
	5.06	2.17	61.5
	4.45	1.71	26.3
Engine Rooms	0.61	0.35	25.0
	21.00	2.40	115.0
	9.58	0.81	7.0
	2.98	0.67	27.0
	1.96	1.37	84.0
Brick Stowage Space	38.59	25.57	309.0
	35.49	15.03	225.6
	35.66	14.34	337.1
	233.30	85.50	895.6
	264.30	100.00	331.0

TABLE 3.N

CALCIUM SILICATE SECTIONS AND ASBESTOS CLOTH. APPLICATION OF PIPE LAGGING

Location	Gravimetric samples mgm/m³		Fibres per c.c.	Location	Gravimetric samples mgm/m³		Fibres per c.c.
	Total dust	Respirable dust			Total dust	Respirable dust	
Boiler Rooms	7.59	1.90	5.00	Engine Rooms	2.76	1.02	3.00
	2.83	0.95	9.00		1.64	0.96	0.80
	16.70	3.78	13.00		2.42	1.11	0.80
	10.67	3.20	11.00		2.34	1.50	0.30
	45.75	1.10	55.00		8.50	0.62	0.10
	8.37	3.45	4.00		2.80	0.30	16.00
	10.94	4.19	41.00		15.54	0.39	0.10
	4.25	0.63	6.00		1.69	0.55	10.90
	4.05	4.78	12.00		0.75	0.36	0.90
	7.56	2.15	9.00				
	0.52	0.26	0.13				
	0.72	0.26	0.18				
Engine Rooms	0.39	0.26	40.00	Accumulator Compartment	11.49	1.04	6.00
	0.45	0.39	9.00		26.62	2.32	6.70
	0.58	0.38	4.00		1.91	0.54	2.70
	1.59	0.38	29.00		3.52	0.64	2.90
	2.80	1.29	1.00		10.14	0.60	15.10
	4.87	1.06	2.00		4.83	0.99	7.19
	3.88	2.68	0.10		5.82	1.10	11.18
	4.79	1.07	0.10		2.25	0.53	6.40
					3.29	1.40	18.70
					1.86	0.65	0.10
					2.18	0.55	1.57
					1.19	0.52	2.30

A CLINICAL, RADIOLOGICAL AND PHYSIOLOGICAL SURVEY OF MEN EXPOSED TO ASBESTOS IN HM DOCKYARD, DEVONPORT

A Clinical, radiological and physiological survey of Devonport Dockyard workers exposed to asbestos dust.

The Subjects

The population at risk has been described earlier in this report and is shown in figure 2.1 page 43. It was decided to examine all the men trained to use asbestos spraying machines; all the full time asbestos workers; and a representative sample of the men considered to be intermittently exposed to asbestos.

It was hoped to avoid the difficult problem of "control" groups by studying this population with a very wide range of exposure to asbestos both in terms of concentration, and duration of exposure.

Table 4.1

Numbers of men selected for examination

Sprayers	55
Laggers	108
Sailmaker Laggers	15
Storemen	19
Masons	25
Intermittently exposed	198
Total	420

The Asbestos Sprayers

This process ended in 1963 so that the seventy-one men who were trained to use the machines were no longer employed on this work. Twelve had died, and four could not be traced, leaving 55 men as subjects for this study. Thirty-six of these still worked in the Dockyard and the remaining nineteen had either retired at the age of 65 or had left the Dockyard

for work elsewhere. (See footnote*).

Full time asbestos workers

Laggers

There were 108 laggers employed on ships and in the asbestos mattress shop.

Sailmaker Laggers

Fifteen men were employed using asbestos cloth, and other materials to insulate cold water pipes and ventilation trunking.

Asbestos Storemen

Nineteen men were employed in asbestos stores.

Masons

Twenty-five men were employed as masons using asbestos cement or applying and grinding terrazo flooring which contained a small amount of asbestos.

Men intermittently exposed to asbestos

The employing departments were asked to submit the names of men considered to be occasionally exposed to asbestos dust. In July 1966 a list of 1,992 names was produced and from this list a random sample of 198 men were selected for examination. Table 4.II shows the numbers of men in various occupational groups thus selected.

*The causes of death for the 12 sprayers who have died are given in the Appendix to this section (page 243).

Table 4.II

Numbers of men in intermittently exposed occupational groups

Skilled labourers afloat	51
Skilled labourers ashore	8
Sailmakers afloat	3
Joiners afloat	6
Plumbers afloat	9
Masons afloat	3
Painters afloat	26
Shipwrights afloat	14
Boilermakers afloat	10
Welders ashore and afloat	11
Burner, riveter, caulker, driller	6
Engine fitter afloat	18
Electrical fitter afloat	16
All other Dockyard occupations	17
Total	198

Methods

Each man in the survey was sent a letter which explained the need for research into asbestosis, the form of the medical examinations, and asked for his co-operation.

A great deal of time was spent in persuading those men who "forgot", "could not be bothered", or who "did not see the point of the tests", to attend for examination.

The procedures for each subject were:-

a. Full size chest x-ray

b. Standard respiratory questionnaire

c. Clinical examination

d. Tests of lung function

e. Examination of sputum for asbestos bodies and fibres

f. Measurement of the degree of finger clubbing.

a. Chest X-ray

Posterior anterior chest radiographs were taken by the Pneumoconiosis Research Unit Mobile X-ray Team, using a standardized technique and automatic film processing. The films were read by the author each evening in order to make a preliminary classification, and also to detect other diseases which might need further investigation and treatment. Four men were suspected of having active pulmonary tuberculosis and were referred to the local chest clinic. Three of these were found to require treatment.

For the purpose of the survey the films were read independently by a panel of four readers. The method of classification was that proposed at Cincinnatti in 1963. This was a development of the 1958 Geneva classification with additional symbols for recording irregular opacities, and the type, extent and site of pleural changes. An example of a standard reading sheet appears in the appendix to this section, (page 253).

The panel members were:-

Dr. J.C. Gilson, Director, Pneumoconiosis Research Unit, Penarth

Dr. G.K. Sluis-Cremer, Miners Medical Bureau, Johannesburg, S. Africa

Professor J.C. McDonald, McGill University, Canada

Dr. P. Cartier, Quebec Mining Company, Quebec, Canada.

b. } Respiratory symptoms, occupational history and the findings of the
c. }

clinical examination were recorded on the Medical Research Council long Respiratory Questionnaire (1960)*. All the interviews and clinical examinations were carried out by the author. Physical signs were recorded in a standard manner, and included the following observations:-

Assessment of general physique.

Quality of the heart sounds; presence of any murmurs.

Pulse rate.

Blood pressure was measured with the patient seated.

Presence or absence of cyanosis.

The position of the trachea, and assessment of chest movement.

Measurement of chest expansion.

Presence or absence of rales or crepitations.

Presence or absence of rhonchi.

Assessment of finger clubbing - nil; early or doubtful; positive.

Presence or absence of asbestos corns.

The occurrence of any other condition having an effect on lung function, e.g. severe kyphoscoliosis; thoracoplasty, etc.

The subjects were measured standing without shoes; sitting for stem height. They were weighed without shoes or top coats.

A sample of venous blood was obtained during the clinical examination for estimation of haemoglobin content. This estimation was done at the Pathology Department, Royal Naval Hospital, Plymouth, using the cyanmethaemoglobin coloremetric method.

*See Appendix pages 244-252.

d. Tests of Lung Function

Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second ($FEV_{1.0}$) were measured using the McDermott dry spirometer. (McKerrow et al (1960), Collins et al (1964)). This apparatus has a low inertia and low resistance to gas flow. It gives a linear response and records volume accurately to within 50 ml over the whole range. The timing mechanism is accurate to within $\pm$ 1% and is calibrated daily.

The measurements were performed with the patient seated. The requirements of the test were explained to the subject and the mean of three technically satisfactory blows after two practice attempts was recorded. The results were expressed in litres (BTPS).

Peak Flow Rate (PFR) was measured using the Wright Peak Flow Meter (Wright and McKerrow (1959)). The subject was seated and the mean of three technically satisfactory blows after two practice attempts was recorded. This instrument was not calibrated daily. The results were expressed in litres per minute (BTPS).

Exercise Ventilation (EV) and Standardized Ventilation (SV) were measured using the standardized exercise step test devised by Hugh-Jones and Lambert (1952). A modified gas meter with low resistance to gas flow (0.8 cm of water at 100 litres/min.) was connected to a mouthpiece by means of rubber tubing and two one-way valves.

The requirements of the test were explained to the subject who was seated, given the mouthpiece and fitted with a nose clip. Volume readings were recorded every half minute for at least 3 minutes of rest, (in some cases 5 minutes). The subject was then asked to step on and off the platform in time to a metronome for 5 minutes, and then to sit until the volume readings showed

that recovery had taken place. Readings were made every $\frac{1}{2}$ minute during the exercise and recovery periods. The rate and height of stepping were adjusted to a workload of 350 Kg/min using the nomogram of Cotes (1965).

Those subjects who could not complete the exercise period were told to sit when they had reached the limit of their endurance, and half-minute volumes were recorded until complete recovery had occurred.

Exercise ventilation is the steady state ventilation recorded during the 5th minute of exercise.

Standardized ventilation is the sum of the resting ventilation and the excess exercise ventilation per minute of exercise.

The results are expressed in litres/minute corrected to BTPS.

An example of the calculations used for these tests appears in the Appendix to this section.

The measurement of lung volumes and transfer factor (CO single breath) and its sub-divisions.

These indices were measured using the apparatus developed at the Pneumoconiosis Research Unit (Cotes (1965), Meade et al (1965)). The apparatus was set up, tested for leaks and the dead spaces of the instrument calibrated. (Cotes (1965)).

Helium was estimated using a Cambridge Katharometer calibrated for helium in dry, CO_2 free air. Calibration of the instrument showed a slightly non-linear response and a small correction factor was calculated. The effect of oxygen and nitrogen on the instrument was obtained by calibration, and a correction factor calculated for use during experiments involving gas with high oxygen content. The instrument was calibrated twice daily, to compensate for small changes due to atmospheric variation.

Carbon monoxide was estimated using a Grubb Parsons infra-red gas analyser. The instrument was calibrated twice daily.

Oxygen was estimated using a paramagnetic analyser (Servomex Controls Ltd.) which was calibrated daily.

Expired gas was passed through soda lime and self-indicating silica gel to remove carbon dioxide and water vapour before reaching the analysers. An allowance was made in the calculations for the resulting reduction in volume of the samples.

Lung Volumes. The closed circuit apparatus was used to measure the Functional residual capacity by means of the helium dilution method (Gilson and Hugh-Jones (1949), Cotes (1965)). After helium equilibrium was reached the subject was asked to make a forced expiration to residual volume and then a full inspiration to total lung capacity. These procedures were repeated.

The sub-divisions of Total lung capacity measured in this way were:-

Total lung capacity	TLC
Inspiratory capacity	IC
Functional residual capacity	FRC
Expiratory reserve volume	ERV
Residual volume	RV
Vital capacity	VC

The results are expressed in litres BTPS.

Transfer factor (T_L) for carbon monoxide; Diffusing capacity of the alveolar membrane (Dm) and Volume of blood in the alveolar capillaries (Vc)

These procedures were carried out by one technician. The indices were measured using the single breath method described by Forster et al (1954) modified by Cotes (1965), with the same apparatus that was used to measure the lung volumes.

The transfer factor was measured at two different oxygen tensions in order that values for Dm and Vc could be calculated in addition to T_L.

The inspired gas for the measurement at high oxygen tension contained 12-14% helium and 0.2-0.3% carbon monoxide, in oxygen. The gas for the low oxygen tension experiment contained 12-14% helium, 0.2-0.3% carbon monoxide, in air.

The patient was seated and allowed to rest while the requirements of the test were explained to him. The height of the seat was adjusted so that he could breathe easily through the mouthpiece in a comfortable, upright position.

The back tension of carbon monoxide in the blood was estimated for subjects who were smokers. The patient breathed 100% oxygen from a cylinder for 5 minutes and then rebreathed from a 6 litre bag containing oxygen. Carbon dioxide was absorbed by soda lime and after 4 minutes it was assumed that equilibrium was reached between the carbon monoxide in the alveoli and in the bag. The gas in the bag was then analysed for oxygen and carbon monoxide.

The transfer factor at high oxygen tension was then estimated. The subject was instructed to exhale until he reached his residual volume. He then inhaled the test gas to within 200-400 ml of his total lung capacity. The volume to be inspired was calculated from the previous measurement of his vital capacity. The breath was held for 10 seconds, and during expiration a sample of 600-800 ml of alveolar gas was collected for sampling, after the

first 600-300 ml of gas was allowed to escape to flush out the dead space. The volume inspired and the volume of the sample was calculated from the tracing, and the breath-holding time was taken to begin after 1/3 of the time of inspiration and to end at half the time of sample collection. (Jones and Meade 1961).

After an interval of 10 minutes, during which time the apparatus was prepared, the experiment was repeated using the inspirate gas mixture with low oxygen content.

The alveolar volume during breath-holding was calculated by adding the volume inspired to the residual volume estimated by the helium dilution method.

The effective alveolar volume (Cotes 1965) was calculated from the dilution of helium in the test gas during the breath-holding experiment.

The indices calculated from these experiments are:-

Alveolar volume Va litres BTPS

Effective Alveolar volume Va^1 litres BTPS

Transfer factor CO
Transfer factor CO (10 sec Va^1) $T_L{}^1$ } ml CO/min/mmHg

These two indices were calculated at low and high oxygen tensions.

Transfer Coefficient KCO. This is the ratio of T_L/Va expressed in ml CO/min/mmHg per litre of alveolar volume BTPS.

Diffusing capacity of the membrane Dm

Alveolar capillary volume Vc

These two indices were also calculated using the effective alveolar volume (Va^1 10 sec) and are designated Dm^1 and Vc^1.

The data obtained from the spirometer tracings and the gas analysers were recorded on a data sheet. The numerical data were then punched on to tape and

processed in an electronic computer. The results of the calculations were printed out on the reverse of the data sheet enabling a check of the accuracy of data recording, and punching in the event of apparently incorrect or unacceptable results.

The basic calculation, together with examples of data sheet and computer print out are to be found in the Appendix to this section.

The programme for the computation was prepared by Mr. B. Sweet of the Plymouth College of Technology from the standard calculation provided by the author.

e. Examination of sputum for asbestos bodies and fibres

Each subject was issued with one sputum bottle at his attendance for chest x-ray. He was instructed to spit into it all the phlegm produced in the first hour after rising from bed. He was told to return the bottle even if he could not produce sputum.

Two further bottles were issued at his attendance for medical examination. He was told to return one the next day, and to keep the other until he had a chest cold with sputum before returning it.

An equal volume of fresh eusol solution was added to the returned bottles that contained sputum and a wet preparation of the centrifuged deposit was examined under transmitted light for asbestos bodies and fibres. One technician of the Department of Pathology, Royal Naval Hospital, Plymouth, was responsible for all these examinations.

f. Preparation of finger casts for measurement of hyponychial angle

Casts of the index finger of each subject were prepared using the technique described by Regan et al (1967). Where the index fingers of both hands were unsuitable due to injury or other deformity the middle finger was used.

Measurement of the hyponychial angle was made by one technician, the points for this measurement being marked on a shadow of the cast thrown by a parallel beam of light. The value for this angle was recorded with the other clinical data.

Sequence of procedures

The chest x-ray examinations were made over a three week period in October 1966. Men who did not attend at that time were examined as they were traced over the following two years.

Medical and physiological examinations proceeded over $1\frac{1}{2}$ years. An average of 5 subjects were seen daily when the equipment was working satisfactorily, but several delays were caused by mechanical failure of the apparatus, and by defects in the carbon monoxide gas analyser.

At examination the subjects were first weighed and measured and the respiratory questionnaire was completed. The tests of ventilatory capacity (FVC, $FEV_{1.0}$ and PFR) were then performed, and this was followed by the measurement of lung volumes, transfer factor and its sub-divisions. The exercise ventilation was next measured, and the examination was completed by the making of a finger cast. Each complete examination took about $1\frac{1}{2}$–2 hours to complete.

All the interviews, clinical examinations, tests of ventilatory capacity and exercise ventilation were undertaken by the author. One technician performed all the measurements of lung volume, T_L, Dm and Vc.

The apparatus was installed and calibrated and after gaining experience in its use a repeatability trial was carried out. Twelve normal subjects were asked to co-operate and all the measurements of lung volume, transfer factor and its sub-division were made on them on two occasions. Ten subjects had tests of ventilatory capacity and exercise ventilation measured on two occasions.

The reproducibility of a single measurement for each test including both within day and between day variability is shown in Table 4.III. The data from these tests are to be found in the Appendix to this section.

Table 4.III

Repeatability lung function tests

Test	Mean	Standard Deviation	Coefficient of Variation %
*$FEV_{1.0}$	3.56	0.06	1.13%
*FVC	4.32	0.10	1.57%
*Exercise Ventilation	33.7	2.88	5.74%
*Standardized Ventilation	33.1	2.98	4.27%
°Vital capacity	4.63	0.28	4.16%
°Residual Volume	1.34	0.17	8.60%
°Total Lung capacity	6.02	0.24	2.92%
°Alveolar Volume	5.60	0.19	2.32%
°Effective (10 sec) Alveolar Volume.	5.52	0.98	3.81%
$^{o}T_L$	28.7	0.95	2.41%
$^{o}T_L^1$ (using 10 sec effective Alveolar volume).	28.2	1.17	2.87%
°Dm	48.2	6.32	9.29%
°Vc	67.5	11.77	11.85%

*Estimations on 10 subjects on two occasions.

°Estimations on 12 subjects on two occasions.

The Results

The data were recorded numerically and transferred to punched cards. The data relating to the questionnaire, clinical findings, and lung function results were punched on three cards for each subject, and another four cards contained the x-ray reading for each man.

Computer programmes were written by Mr. E.C. Tapper for analysis of this data to the requirements of the investigator, and the data analysed on a computer at the Plymouth College of Technology. Other tabulations and calculations have been undertaken at the Medical Research Unit by the author.

Table IV gives the numbers of men in each occupational group selected for study together with the numbers of men examined. The overall response rate was 96% with the lowest response rate, 93%, in the intermittently exposed group.

Table 4.IV

Numbers of men in occupational groups with numbers examined.

Group	Number in group	Number examined	% Response
Sprayers	55	52	95%
Laggers	108	107	99%
Sailmaker Laggers	15	15	100%
Storemen	19	18	95%
Masons	25	25	100%
Intermittently exposed	198	185	93%
Total	420	402	96%

The results of the dust sampling survey (pages 84-139) suggested that there were different intensities of exposure to asbestos associated with different occupations. It was not possible in this study to look at each occupation, but it seemed reasonable to consider the occupational groups selected for study as being broadly representative of different levels of asbestos exposure. The sprayers had the highest exposure, followed by the laggers. The intermittently exposed group had the least exposure. The sailmakers, storemen and masons all had roughly equal exposure and it was convenient to consider these as one group. The level of exposure of this combined group would be somewhere between that of the laggers and the intermittently exposed.

Exclusions from Analysis

Table 4.IV refers to all the men examined in the study, but for the analysis of the respiratory questionnaire, chest radiographs, and lung function tests it was decided to exclude those men who had other diseases which might have an adverse effect on lung function. The numbers and reasons for these exclusions are given in Table 4.V. There were 11 men who had been exposed to other dusts during their working lives, and even though most of these exposures were short in comparison with the asbestos exposure it was decided to exclude them from the analysis. The type and length of exposure for these men is shown in Table 4.VI . Data relating to these men are shown in the appendix to this section (pages 240-1), and they will be discussed later in this report.

Population for Analysis

The numbers of men in each occupational group included in the analyses are given in Table 4.VII. The age structure of this population is shown in

Table 4.VIII. The sprayers have a high mean age because there are no young men in this group. (The process was discontinued in 1963). Tables 4.IX and 4.X show the time from first exposure to asbestos and the duration of exposure. The average height of the subjects is recorded in Table 4.XI , and there is little difference between the groups.

Respiratory Symptoms and History of Previous Chest Illness

Table 4.XII shows the number of men in each occupational group with respiratory symptoms and history of previous chest illness, and these numbers are expressed as percentages in figure 4.1 .

Cough and phlegm was considered to be present in those men who stated they had those symptoms on most days for as much as three months each year (Questions 1-10). There are higher proportions of men with productive coughs in the two most heavily exposed groups, the sprayers and laggers, with lower proportions in the sailmakers, storemen and masons, and in the intermittently exposed ($X^2 = 14.74, n = 3, P = > 0.005$).

There are few men who admit to a period of increased cough and phlegm lasting for three weeks or more (Question 12), and any differences between the groups are not evident.

Dyspnoea

Question 14 attempts to grade the degree of dyspnoea into the following six categories:

Grade 0. Those men disabled from walking by any condition other than lung or heart disease.

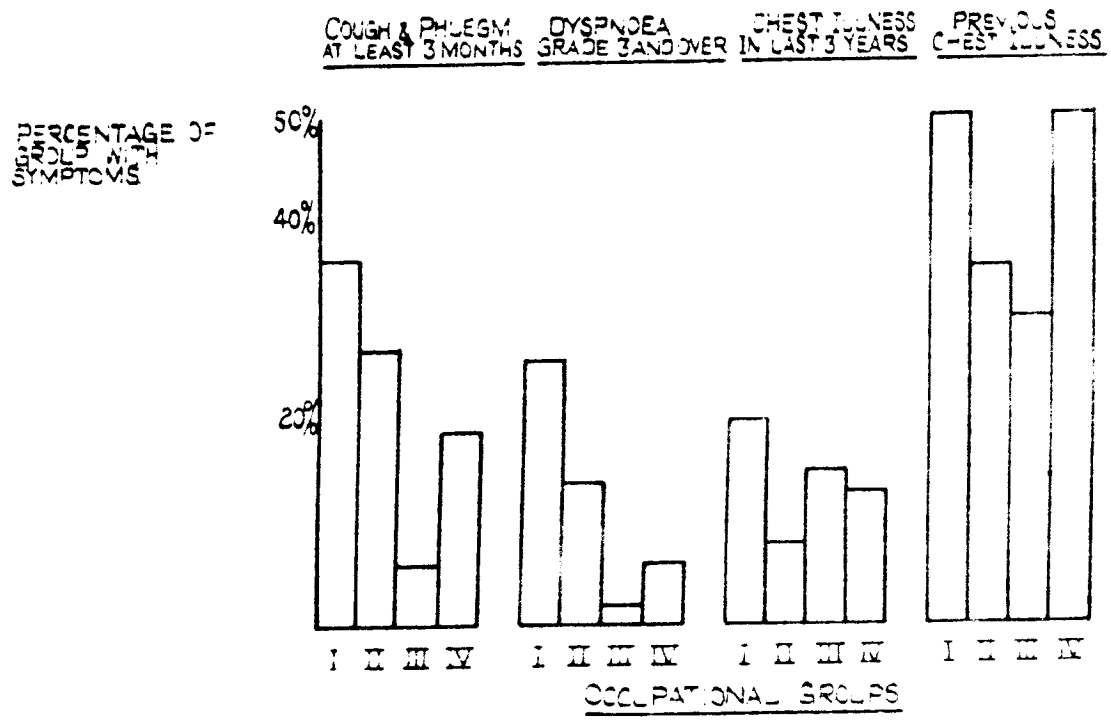
Grade 1. Normal. No breathlessness hurrying on level or walking up a slight hill.

Grade 2. Short of breath hurrying on the level or walking up a slight hill.

Grade 3. Short of breath walking with other people on the level.

FIG 4.1

RESPIRATORY SYMPTOMS AND PREVIOUS CHEST ILLNESS IN OCCUPATIONAL GROUPS



Grade 4. Short of breath walking at own pace on the level.

Grade 5. Short of breath washing or dressing.

Few men were disabled by other conditions (Grade 0). The most common disabling conditions were arthritis, injury or amputation of the lower limbs.

Grade 2 dyspnoea is of doubtful value in suggesting that there is breathlessness on exertion, but there are proportionately more men with Grade 2 dyspnoea in the most heavily exposed group. A more reliable indicator of effort dyspnoea is probably obtained by considering those men with Grades 3, 4 and 5 dyspnoea. There are large differences between the occupational groups for the percentages of men with Grade 3 or more dyspnoea. The sprayers and laggers have the highest proportions of such men; the sailmakers, storemen and masons, and the intermittently exposed have the lowest proportion ($X^2 = 20.79$, $n = 3$, $P = < 0.001$).

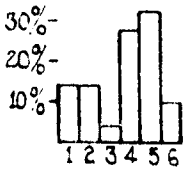
The proportions of men with a history of chest illness in the last three years (Question 21) do not differ very much, but the sprayers again have the highest value. There is no relationship between the history of any chest illness (Questions 22-27) and occupational group. The history of heart disease (Question 28) was recorded for very few men and there does not appear to be much difference between the groups.

Smoking Habits

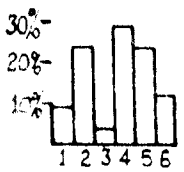
The numbers of men in each occupational group were sub-divided into smoking categories (Table 4.XIII), and these are shown as percentages in figure 4.2. I am not certain how much reliance can be placed on the answers to this part of the Questionnaire (Questions 35-38). I have the impression that there is a tendency for most of the men, especially if they think it will please the questioner, to understate the true amount smoked per day. It may be of interest to record that some men admitted to smoking a certain number of

FIG. 4.2 SMOKING HABITS IN OCCUPATIONAL GROUPS

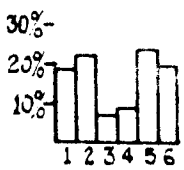
PERCENTAGE IN SMOKING CATEGORIES.



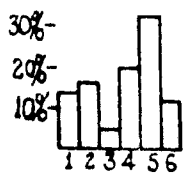
SPRAYERS



LAGGERS



SAILMAKERS, STOREMEN AND MASONS.



INTERMITTENTLY EXPOSED.

1 = NON-SMOKER
2 = EX-SMOKER
3 = 1-4g/day
4 = 5-14g/day
5 = 15-24g/day
6 = 25+g/day

cigarettes per day, but when questioned further they admitted to smoking almost as many again after leaving work. They appeared to consider their "smoking day" to be of the same duration as their "working day" and that what they did after work was another matter.

There are no large differences in smoking habits between the groups, with about 70% of men in each group classified as smokers. The sailmakers, storemen and masons had fewer smokers (60%), and the laggers fewer non-smokers, but more ex-smokers, than the sprayers and intermittently exposed.

Physical Signs

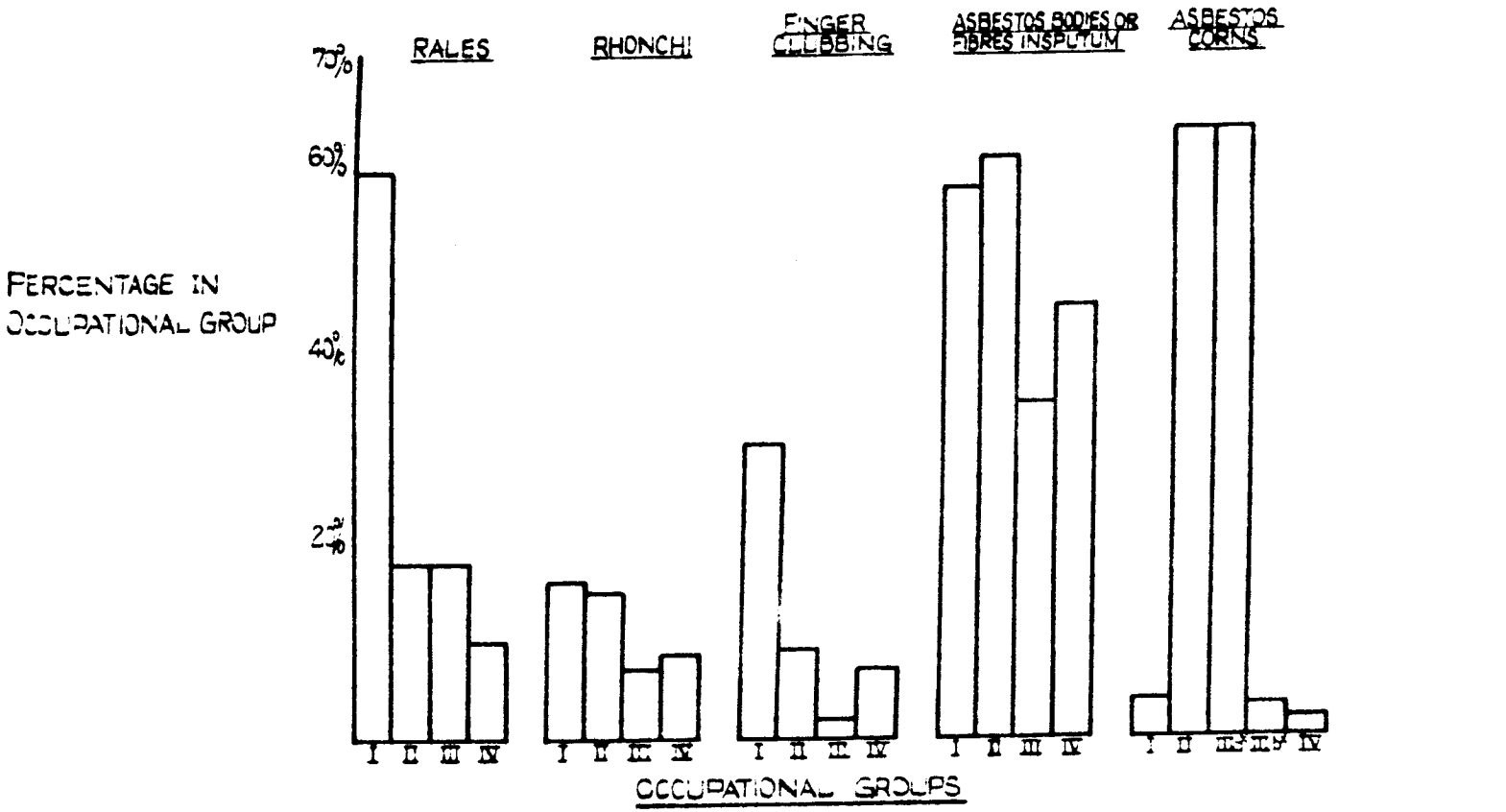
The presence of selected physical signs occurring in the population, and in each occupational group is shown in Table 4.XIV and figure 4.3. Rales were found most frequently in the sprayers and least frequently in the intermittently exposed (X^2 58.36, n = 3, P = 0.001). There was a less noticeable difference in the occurrence of rhonchi between the groups, and although they were found more frequently in the sprayers and laggers than in the groups with less dust exposure these differences were not significant. Finger clubbing was present more often in sprayers and while asbestos bodies or fibres were found in the sputum of high proportions of every group, these proportions were higher in the two most heavily exposed groups (X^2 11.18, n = 3, P = 0.02). Asbestos corns were found to be uncommon except in the laggers and sailmakers, of whom over 60% had these corns on their hands. The mean values for hyponychial angle are shown in Table 4.XV.

Radiographic Abnormalities

The U.I.C.C. classification of chest radiographs was applied to the films independently by the four readers. The profusion of irregular small opacities was graded on the 12 point scale (0/-, 0/0, 0/1, 1/0, 1/1, 1/2, 2/1, 2/2, 2/3,

FIG. 4.3

PHYSICAL SIGNS IN OCCUPATIONAL GROUPS



I = SPRAYERS II = LAGGERS III = SAILMAKERS, STOREMEN & MASONS IV = INTERMITTENTLY EXPOSED

(×IIIa = SAILMAKERS ×IIIb = STOREMEN & MASONS)

3/2, 3/3, 3/4) suggested by Lidell (1963). From the four readings Mr. P.D. Oldham calculated a combined score for the profusion of irregular small opacities for each man.

These scores have been grouped, for the present analysis, into four, corresponding to the elaborated I.L.O. categories 0/0, 0/1, 1, and 2. Each of these groups was sub-divided into those films with, and those without pleural abnormality.

Radiographic Classification

Profusion of small opacities

0/0	Without pleural abnormality
	With pleural abnormality
0/1	Without pleural abnormality
	With pleural abnormality
1	Without pleural abnormality
	With pleural abnormality
2	Without pleural abnormality
	With pleural abnormality

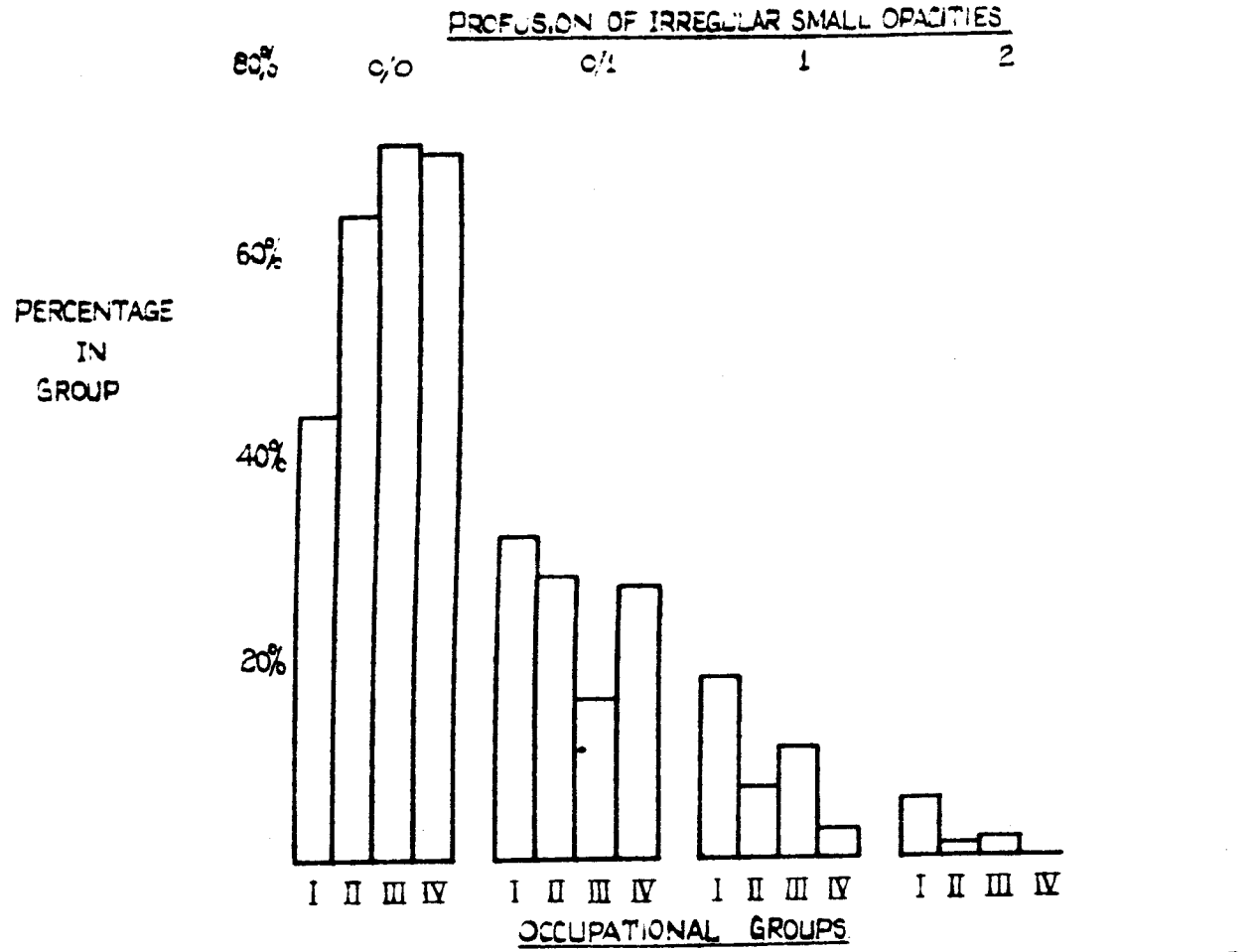
Radiographic Abnormality in Occupational Groups

The numbers and percentages of men in each occupational group sub-divided by x-ray category are shown in Table 4.XVI. Figure 4.4 shows the percentage of men in occupational groups for each category of small opacities regardless of pleural abnormality.

The most heavily exposed groups, the sprayers and laggers, have fewer men with normal films than do the lesser exposed groups ($X^2 = 12.99$, $n = 3$, $P = < 0.005$). In the heavily exposed groups there is a consistent trend showing proportionately more men with abnormal films in each category of radiographic abnormality.

FIG 4.4.

RADIOGRAPHIC CLASSIFICATION IN OCCUPATIONAL GROUP



I = SPRAYERS, II = LAGGERS, III = SAILMAKERS, STOREMEN & MASONS, IV = INTERMITTENTLY EXPOSED.

The percentage of sprayers with pleural abnormality was 48%, laggers 33%, sailmakers, storemen and masons 18%, and intermittently exposed 32%. There was a trend towards more abnormality occurring in the most highly exposed groups ($P = 0.05$).

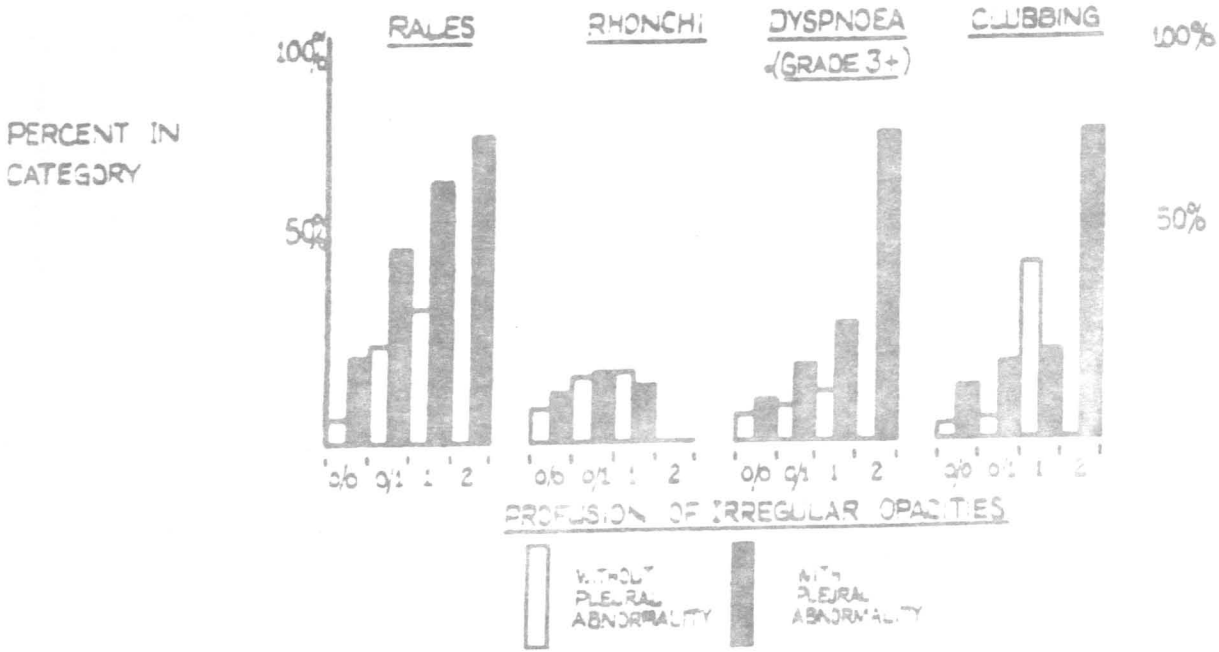
In all these Tables, and for the subsequent data relating to lung function in x-ray categories there is one peculiar result in X-ray Category 2 without pleural abnormality. This tall, healthy young man has a radiograph showing irregular small opacities which cannot be explained clinically or in terms of dust exposure. He is normal in every other respect and has not been exposed to dust other than 6 years recent intermittent exposure to cement and asbestos.

Apart from this one anomalous result Table 4.XVII shows that the mean age of men with pleural abnormalities is greater in each category of opacities than that of men with normal pleura. This Table also shows that the mean years of exposure, and mean years from first exposure increases in those men with increasing radiographic abnormality. The duration of exposure is longer for men with pleural abnormality but normal lung parenchyma than it is for men with completely normal radiographs.

Signs and Symptoms in X-ray Categories

The proportions of men with physical signs, and with Grade 3 or more dyspnoea are seen in figure 4.5. Rales, clubbing and dyspnoea occur more frequently with increasing x-ray abnormality ($P = < 0.001$). There does not appear to be a similar trend for the proportions of men with rhonchi. (Table 4.XVIII).

FIG 4.5 PHYSICAL SIGNS
AND DYSPNOEA IN X-RAY CATEGORIES



Lung Function Tests in Total Population and Occupational Groups

The mean values, standard deviation and regression on age for all the tests of lung function are shown in Tables 4.XIX-XXIII. The values for these tests were adjusted for each subject to the mean age and height of the population using the regression equations in Table 4.XXIV. (Cotes 1965). The means for these adjusted values in occupational groups are shown in figure 4.6. The use of independent regression equations, rather than those derived from the study population, is an attempt to show the possible effect of asbestos exposure on lung function in the respective groups. The internal regression equations will be affected by any effect of asbestos exposure, as well as age and height, while those independently derived will not.

The differences between occupational groups for these lung function tests are small, but they are consistent. There is a reduction in FVC, SVC and TLC in the groups with most intense exposure. Analysis of variance supports the hypothesis that there is a specific between group variation ($P > 0.01$). There are less noticeable differences for the values of $FEV_{1.0}$, but again analysis of variance shows that there are between group differences ($P > 0.05$).

The values for residual volume (RV) show little difference, except that the laggers group have a very low value, this low value seems also to be reflected in the TLC result for laggers. T_L and T_L1 also show decreasing values in the group with increasing intensity of exposure ($P > 0.01$). The values for D_M have not been adjusted for age or height because there are no accepted independent regression equations. I suggest that these adjustments would make little difference to the values for D_M, and certainly would not alter the trend because the groups, except for the sprayers, have similar mean ages and heights. This is probably most easily seen by comparing the observed

values and adjusted values for T_L in occupational groups.

Group	Observed Value T_L	Age and Height Adjusted Value T_L
Sprayers	22.17 mlCO	24.3 mlCO/min/mmHg.
Laggers	26.13 mlCO	25.5 mlCO/min/mmHg.
Sailmakers, etc.	28.58 mlCO	28.3 mlCO/min/mmHg.
Intermittently exposed	29.42 mlCO	29.3 mlCO/min/mmHg.

Lung Function Tests in Smoking Categories

The means for age and height adjusted lung function values in smoking categories are shown in figure 4.7. The observed values are given in Table 4.XXV. Values for all lung volumes are lower for the ex-smokers and smokers than the non-smokers, except for RV which is slightly increased in the heavy smoking groups. The values for T_L, T_L1 and D_M show a big fall from non-smokers to ex- and light (1-4G) smokers, but little alteration thereafter.

Lung Function in Radiographic Categories

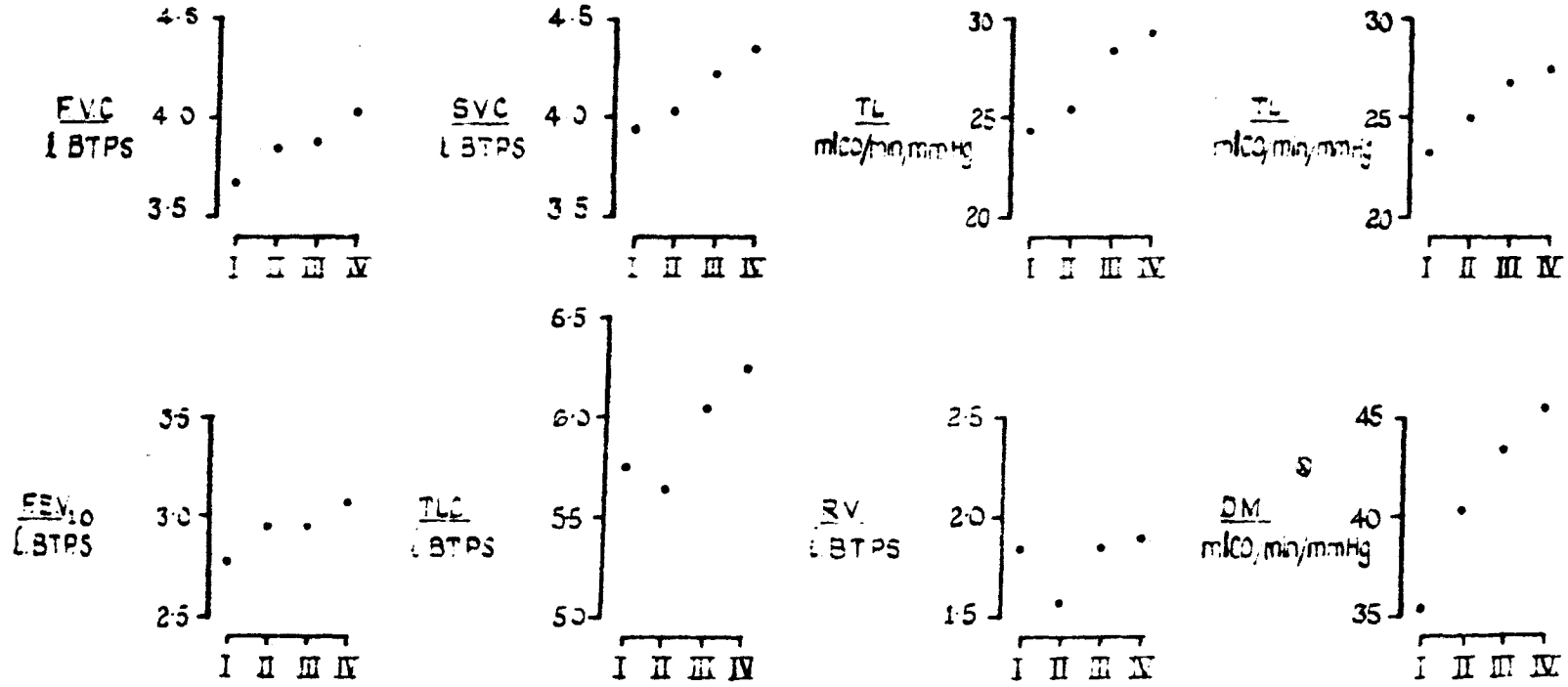
Figure 4.8 shows the mean values for age and height adjusted lung function results in radiographic categories, and the observed values are given in Table 4.XXVI. The differences in the results of these tests are larger than in the two previous analyses and the trends are consistent. Except for Residual Volume (RV) these results show that with increasing radiographic abnormality the values for tests of lung function decrease.

Analysis of variance has been carried out for FVC and shows that there is a definite between group difference ($P = 0.01$).

The values for men with pleural abnormality are all lower than for those without pleural abnormality. The one peculiar result in Category 2 small opacities without pleural abnormality is clearly seen in these results.

FIG 4.6

AGE & HEIGHT ADJUSTED MEAN VALUES FOR LUNG FUNCTION TESTS IN OCCUPATIONAL GROUPS

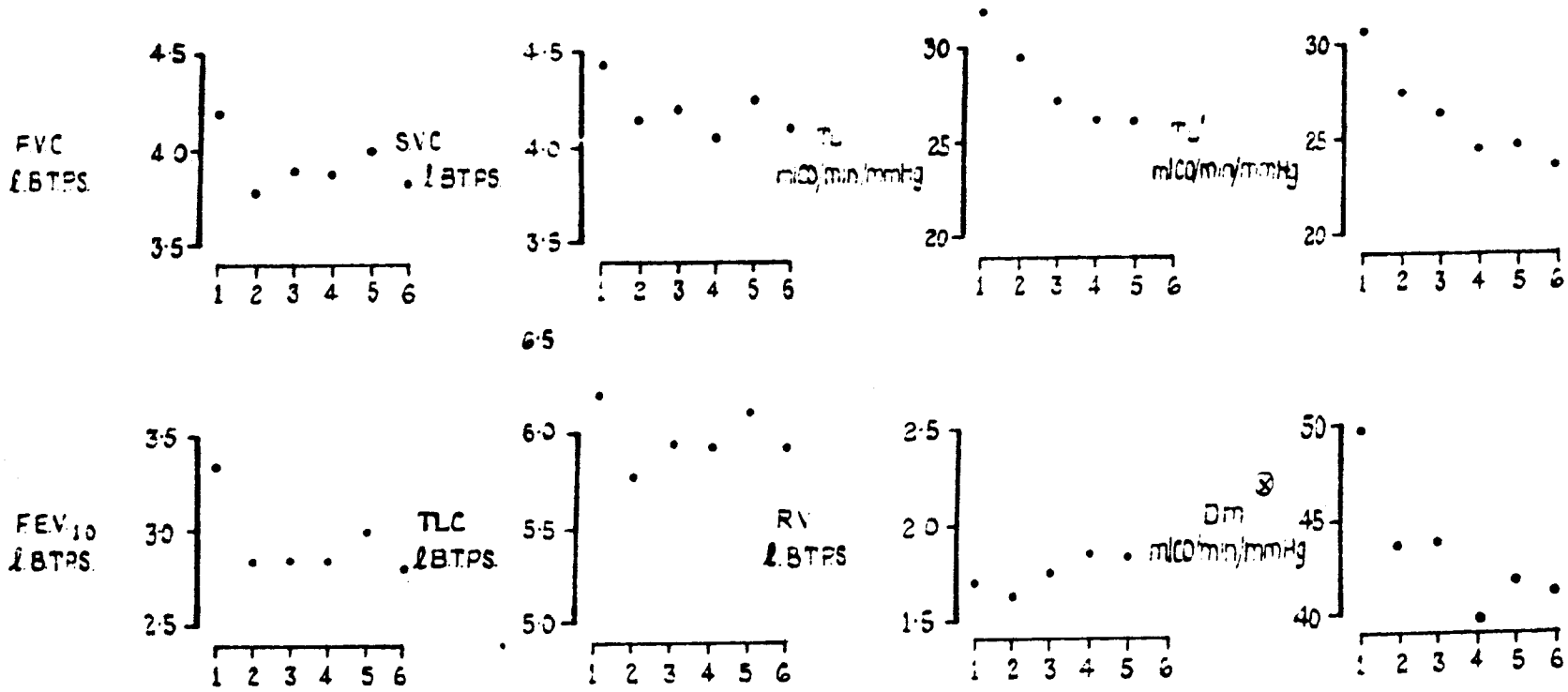


I = SPRAYERS II = LAGGERS III = SAILMAKERS, STOREMEN & MASONS IV = INTERMITTENTLY EXPOSED

⊗ DM NOT ADJUSTED FOR AGE OR HEIGHT

FIG. 4.7

AGE & HEIGHT ADJUSTED MEAN VALUES FOR LUNG FUNCTION TESTS IN SMOKING CATEGORIES

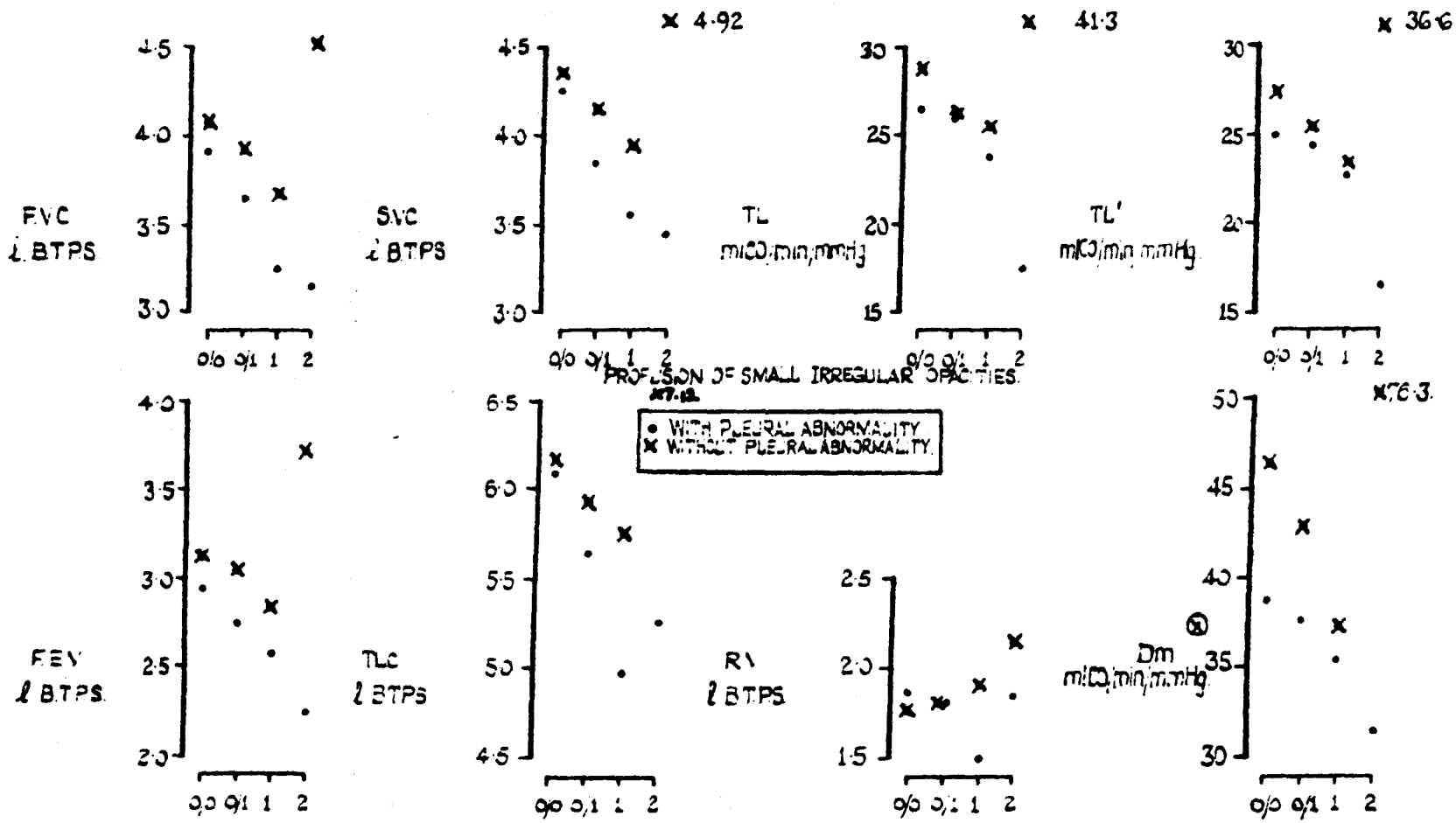


1 = NON SMOKERS. 2 = EX-SMOKERS 3 = 1-4G/day. 4 = 5-14G/day 5 = 15-24G/day. 6 = 25+G/day.

⊗ Dm. NOT ADJUSTED FOR AGE OR HEIGHT

FIG. 4·8

AGE & HEIGHT ADJUSTED MEAN VALUES FOR LUNG FUNCTION TESTS IN X-RAY CATEGORIES.



PROFUSION OF SMALL IRREGULAR OPACITIES

② Dm NOT ADJUSTED FOR AGE OR HEIGHT

Lung Function Assessment

A data sheet containing only the subjects serial number, age, height, weight and lung function results was used to sub-divide these results into patterns of lung function abnormality. Predicted normal values, calculated from the regression equations in Table 4.XXIV, were available for each subject so that allowances could be made for differences in age and height. The subjects were allocated to lung function categories by Dr. J.E. Cotes who had no information about symptoms, signs, asbestos exposure or x-ray abnormality. The subjects were assessed in random order to avoid introducing bias due to the occupational grouping of the men.

The categories were: Normal; Transfer Factor (T_L) reduced; Restriction of Total Lung Capacity (TLC); Transfer Factor reduced and Restriction of Total Lung Capacity; Airways Obstruction.

Lung Function Assessment in Occupational Groups

The numbers and percentages of men in each category are shown in Table 4.XXVII and figure 4.9. There are fewer normal assessments in the sprayers, the highest proportion of normal assessments being found in the intermittently exposed ($X^2 = 30.4$, $n = 3$, $P = < 0.001$). The differences between the groups are small, but there is a consistent trend showing that proportionately more men with abnormal lung function patterns are found in the occupational groups with increasing asbestos exposure. The exception to this is that airways obstruction does not appear to be associated with differences of occupational group.

Signs and Symptoms in Lung Function Categories

The physical signs occurring in the men in different lung function categories are seen in figure 4.10. There are no men with rales in the group with airways obstruction. Basal rales were recorded in 40% of men with reduced T_L;

50% with restricted TLC; and 50% of men with reduced T_L and restricted TLC (T_L + TLC). Rhonchi were recorded in 45% of the men with airways obstruction, in only 11% of those in the normal category and in 8% or less in the remaining groups. There is little difference in the proportions, about 27% of men with dyspnoea in any of the abnormal lung function groups. Finger clubbing was not seen in any of the men with airways obstruction, in only 5% of those thought to be normal, 12% of those with restricted TLC, but it occurred in 40% of those with reduced transfer factor and 31% of those with reduced transfer factor and restricted TLC.

Radiographic Abnormalities in Lung Function Categories

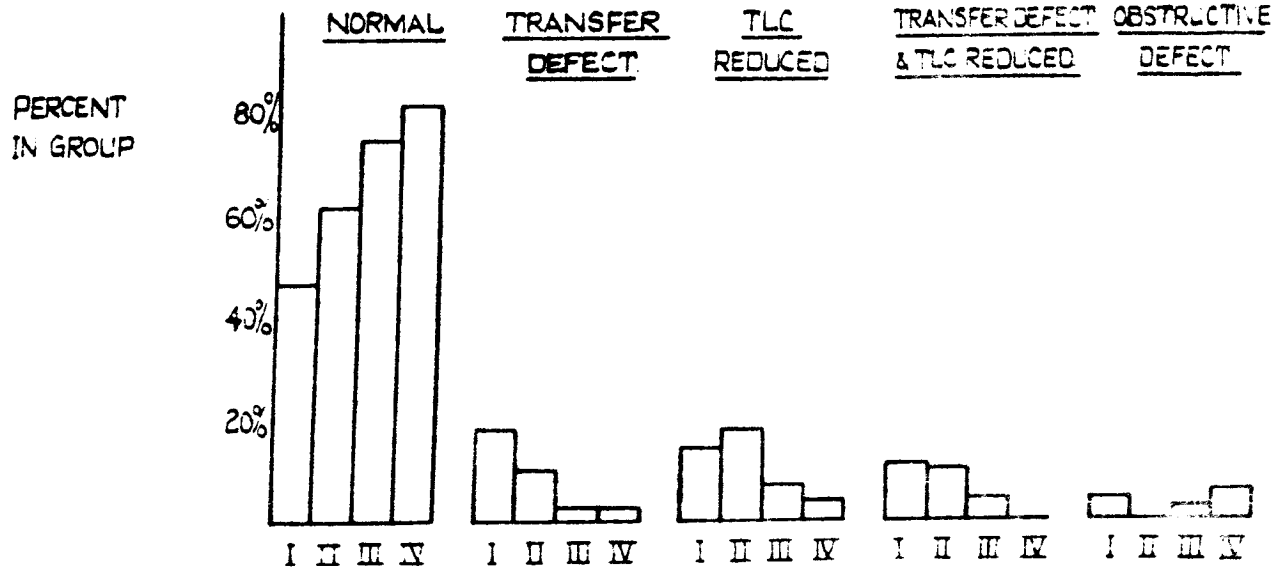
The proportion of men in x-ray categories for each lung function group are shown in figure 4.11. (Table 4.XXVIII). Over 70% of the radiographs were normal for men in the normal, and obstructive lung function groups. In the latter category there were no men with category 1 or 2 radiographs. There were fewer men with normal x-rays, and proportionately more men with abnormal x-rays in the three remaining lung function groups.

Physical Signs and Duration of Exposure

The numbers of men in each occupational group with physical signs and more than grade 3 dyspnoea in 10 year periods of exposure are shown in Table 4.XXIX (figure 4.12). From the figure it can be seen that sprayers have relatively high proportions of men with rales, clubbing, and dyspnoea in all periods of exposure. For the three remaining occupational groups the percentage of men with rales increases with the duration of exposure, and it can be seen that the intermittently exposed have the smallest percentages in each period of exposure. Similar trends are seen for clubbing and dyspnoea, but not for rhonchi.

FIG. 4.9

LUNG FUNCTION ASSESSMENT BY OCCUPATIONAL GROUP

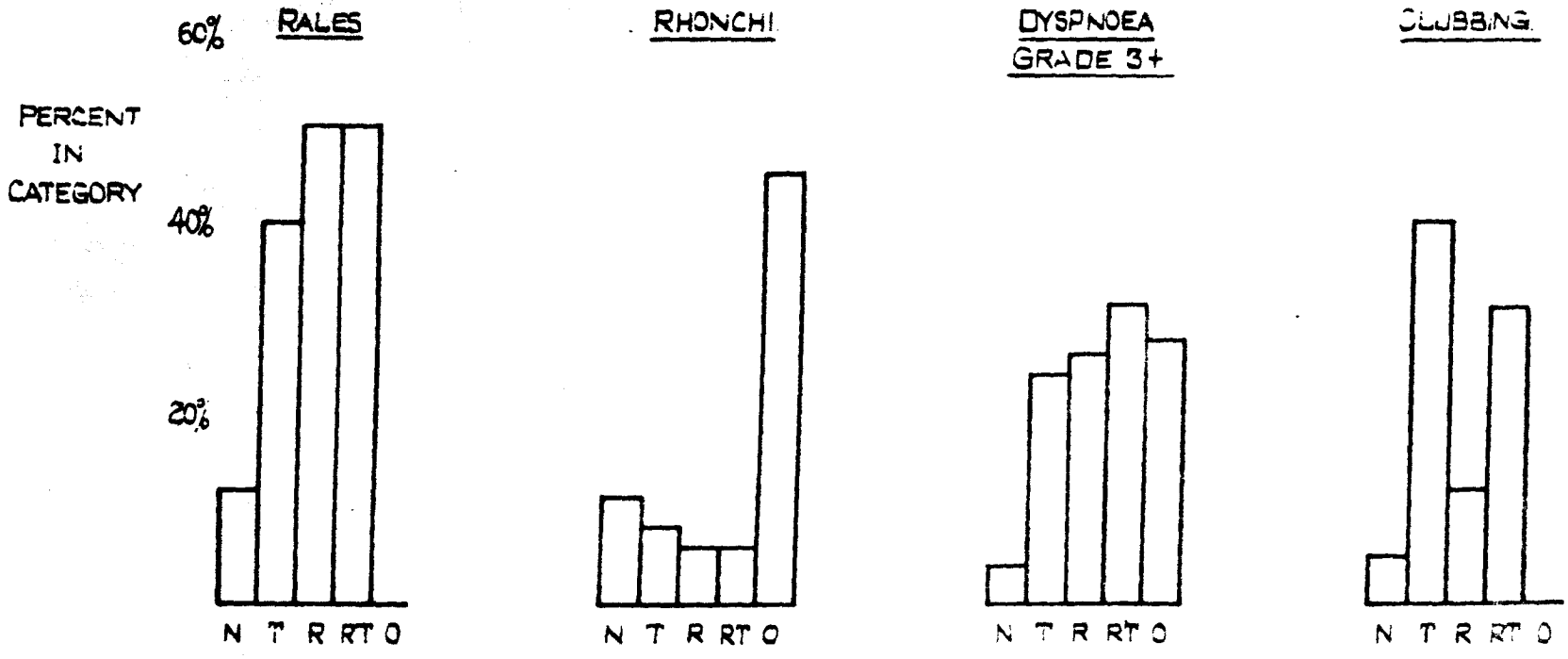


I = SPRAYERS; II = LAGGERS; III = SAILMAKERS, STOREMEN & MASONS;

IV = INTERMITTENTLY EXPOSED

FIG. 4.10

PHYSICAL SIGNS IN LUNG FUNCTION CATEGORIES

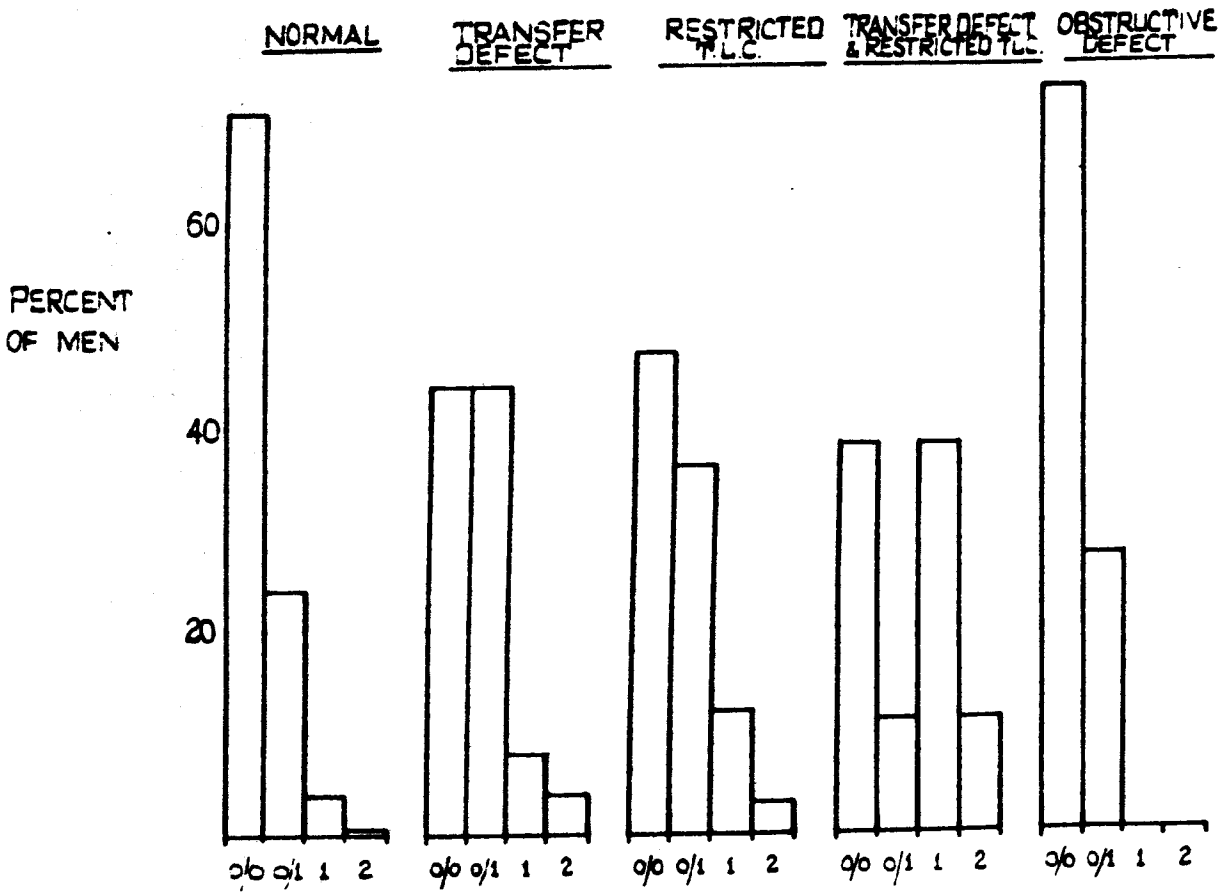


N = NORMAL ; T = TRANSFER DEFECT; R = TLC REDUCED, RT = TRANSFER DEFECT + TLC REDUCED

O = OBSTRUCTIVE DEFECT

FIG 4.11

PERCENTAGE OF MEN IN LUNG FUNCTION GROUPS WITH X-RAY ABNORMALITIES.



The percentages of men from all occupational groups with rales in the four ten year periods are, 0-9 years 3.5%; 10-19 years 25.5%; 20-29 years 32.9%; and 30+ years 34.3% (X^2 = 40.59, n = 3, P = <0.001). For rhonchi the percentages are 8.4%, 12.3%, 16.5% and 11.4%.

Radiographic Abnormality and Duration of Exposure

Table 4.XXX (figure 4.13) shows the number of men in radiographic categories for each occupational group and 10 year periods of exposure. The numbers of men in categories other than Category 0/0 are very small for each occupational group so that they may not be very critically examined. The results for Category 0/0 are useful in demonstrating that there are proportionately fewer men with normal films as the period of exposure increases. For the four occupational groups the percentages of normal films in the four ten year periods of exposure are 0-9 years 83.2%; 10-19 years 60.4%; 20-29 years 47.1%; and 30+ years 48.6% (X^2 = 38.06, n = 3, P = <0.001). They also show that the decrease in the proportion of men with normal films is most severe in the sprayers and laggers, and least severe in the intermittently exposed.

Lung Function Category and Duration of Exposure

The men in lung function categories for 10 year periods of exposure are shown in Table 4.XXXI (figure 4.14). There are more men classified as normal for each period of exposure in the intermittently exposed and fewer such men in the sprayers group. Although the numbers are small in the abnormal categories for the occupational groups, there are trends suggesting increasing proportions of men with abnormal categories with increased duration of exposure. There are more men with abnormal categories in the sprayers and laggers, with fewer in the sailmakers and intermittently exposed. The exception to this trend is shown for those in the obstructive category.

FIG. 4. 12

PHYSICAL SIGNS BY OCCUPATIONAL GROUP AND YEARS OF EXPOSURE

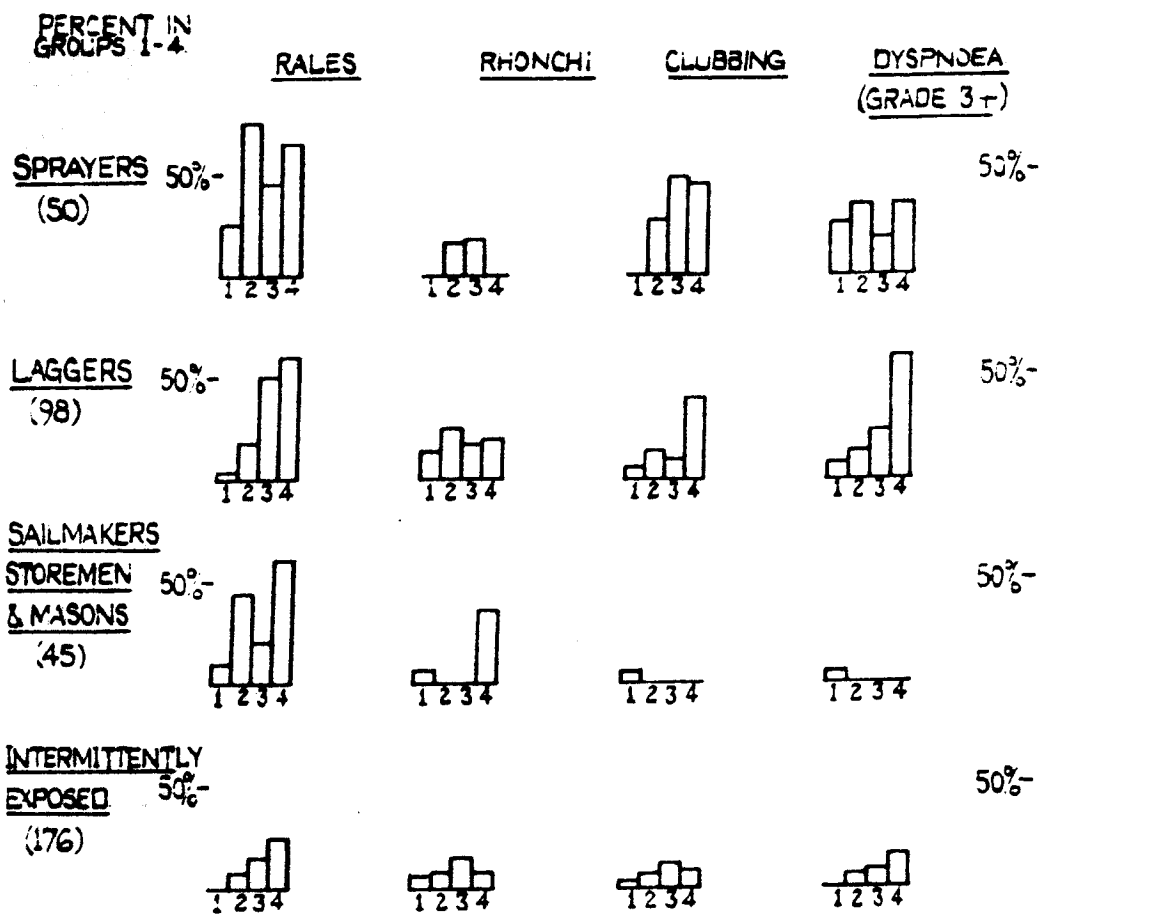
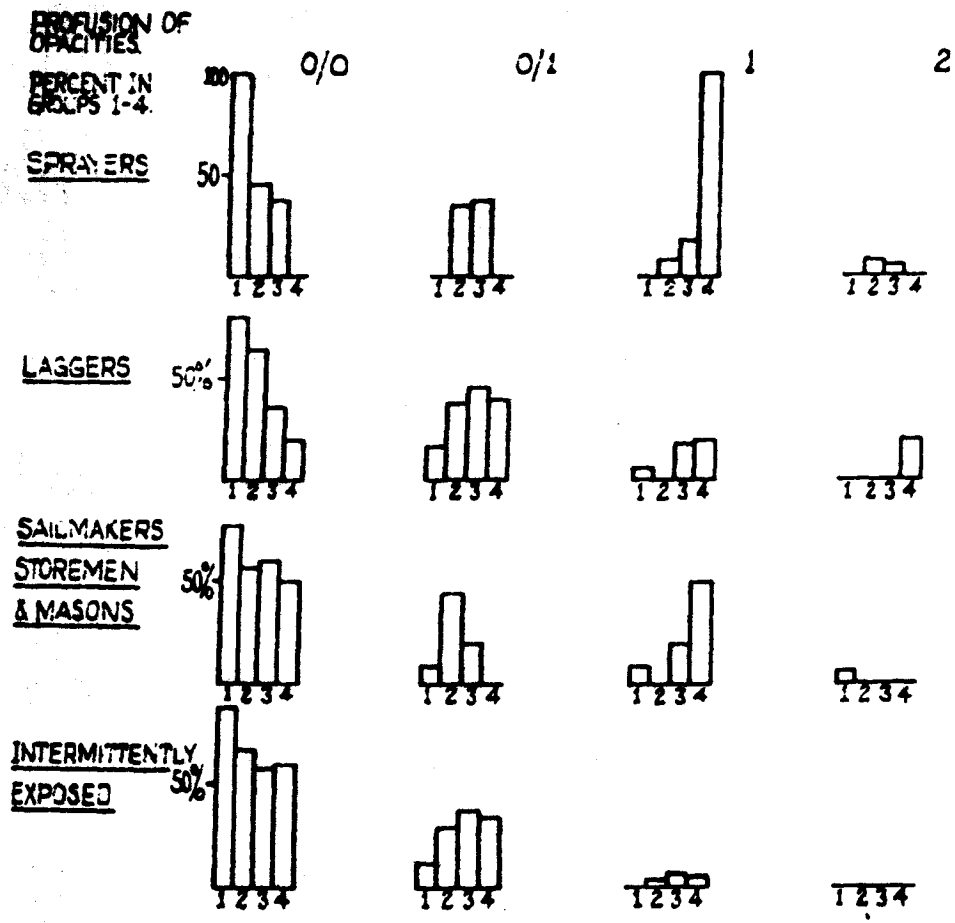


FIG 4.13

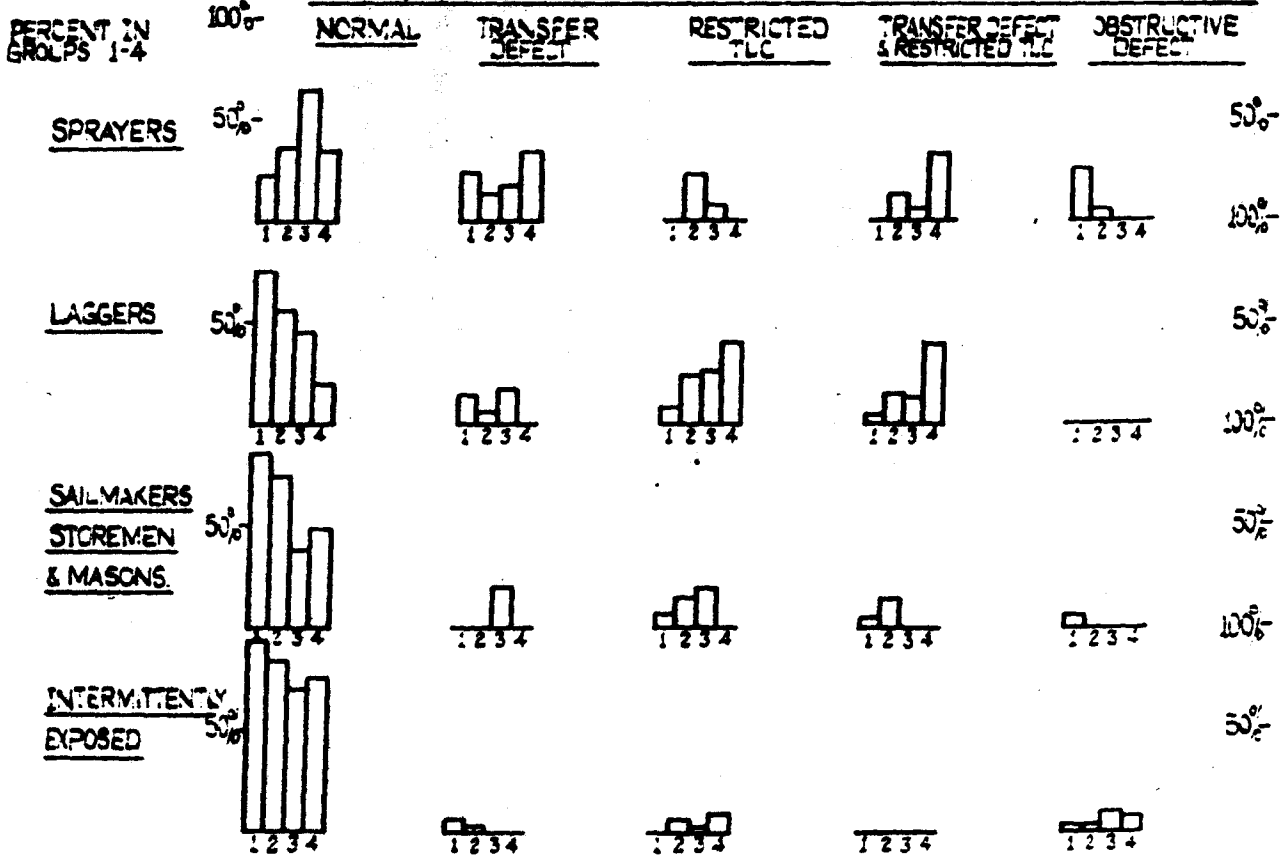
X-RAY ABNORMALITY BY OCCUPATIONAL GROUP AND YEARS OF EXPOSURE



1 = 0-9 years, 2 = 10-19 years, 3 = 20-29 years; 4 = 30 + years of exposure

FIG 4.14

LUNG FUNCTION CATEGORY BY OCCUPATIONAL GROUP AND YEARS OF EXPOSURE.



1 = 0-9 years, 2 = 10-19 years, 3 = 20-29 years, 4 = 30+ years of exposure.

Lung Function Results and Duration of Exposure

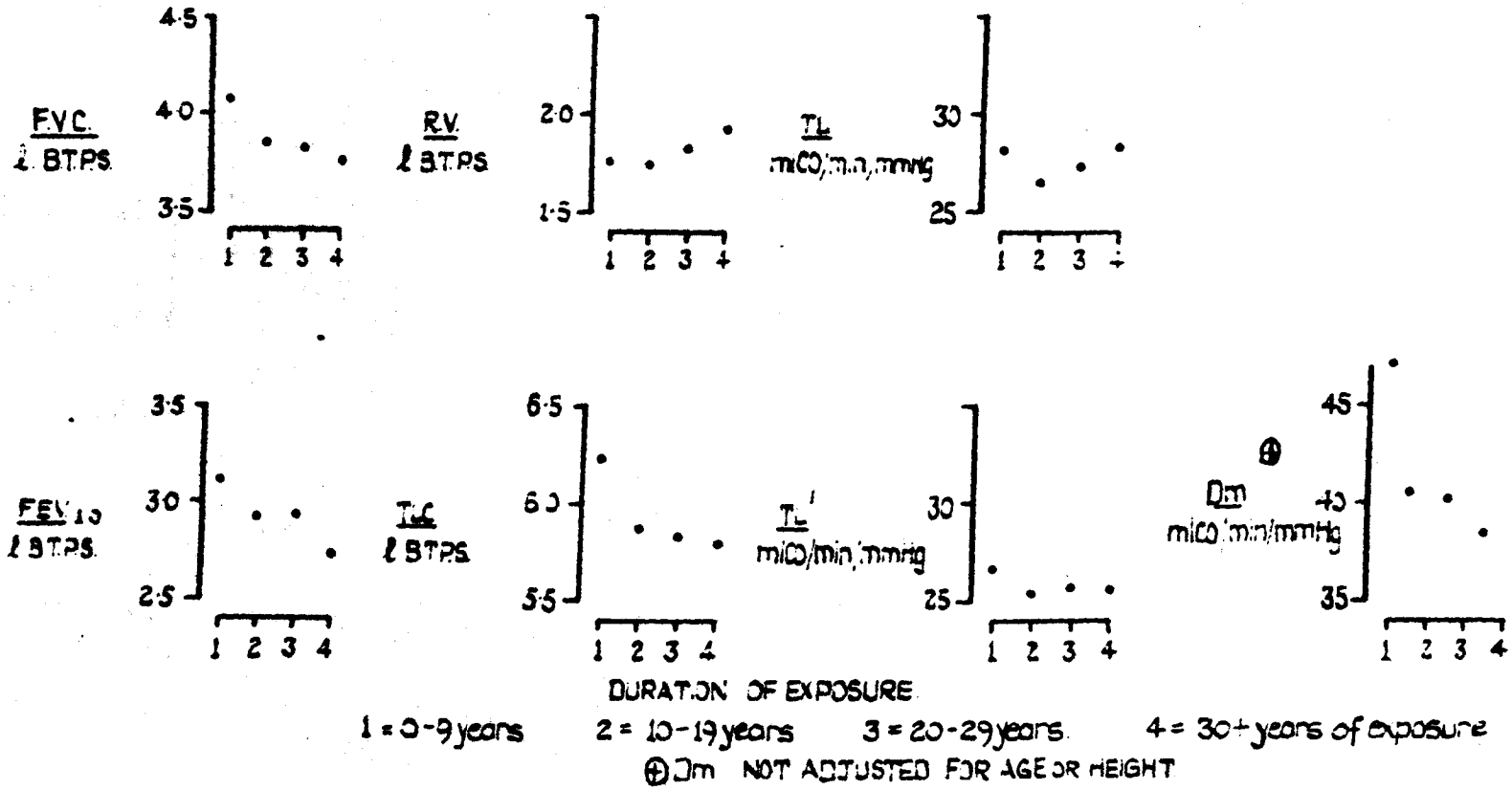
Table 4.XXXII shows the mean values for lung function tests in 10 year periods of exposure for the total population. These values, adjusted to the mean age and height of the population are shown in figure 4.15. It is suggested that FVC, FEV and TLC decrease with years of exposure and that RV shows some increase. T_L shows a decrease in the 10-19 year group and then an increase in the following two periods of exposure. T_L1 shows little change. D_M has not been adjusted for age.

These results may be misleading because the groups with long exposure are heavily weighted by men from the intermittently exposed group, and from the earlier data we know that this group contains large numbers of men with normal x-rays and no abnormal signs. The numbers of laggers (98) and intermittently exposed (176) are large enough to contain sufficient men in the sub-division of years of exposure to make a reasonable comparison possible. The mean values are shown in Table 4.XXXIII, and they have been adjusted for height and age in figure 4.16.

These results suggest a more definite reduction in FVC and $FEV_{1.0}$ with increasing duration of exposure. T_L and T_L1 decrease slightly with increasing duration of exposure in the laggers, but not in the intermittently exposed group. D_M has not been adjusted for age.

FIG 4·15

AGE AND HEIGHT ADJUSTED MEAN VALUES FOR LUNG FUNCTION TESTS IN YEARS OF EXPOSURE 369 MEN



DURATION OF EXPOSURE

1 = 0-9 years 2 = 10-19 years 3 = 20-29 years 4 = 30+ years of exposure

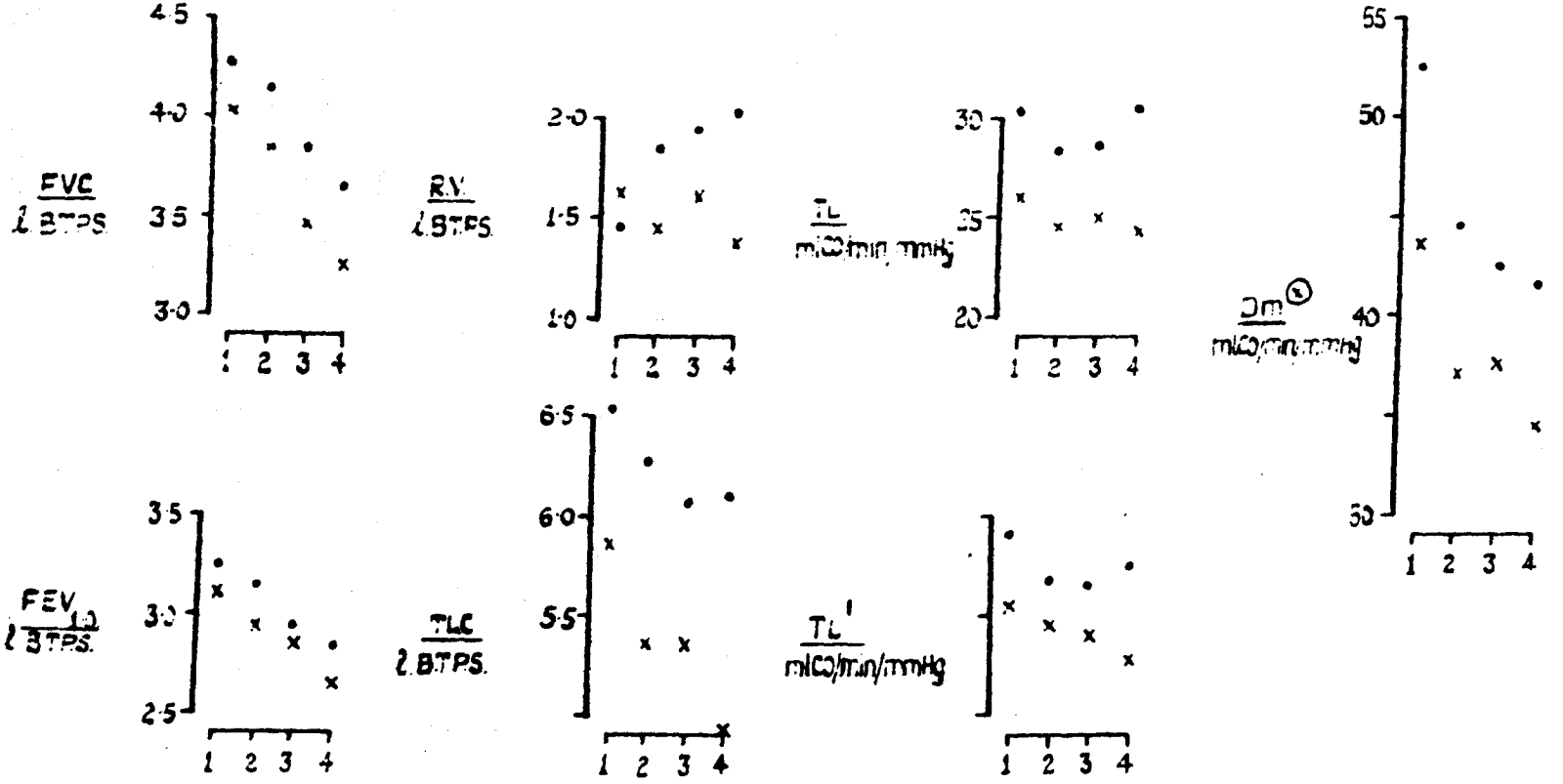
⊕ D.m NOT ADJUSTED FOR AGE OR HEIGHT

FIG 4 16

AGE AND HEIGHT ADJUSTED MEAN VALUES FOR LUNG FUNCTION TESTS BY YEARS OF EXPOSURE ADJUSTED FOR AGE AND HEIGHT.

• INTERMITTENTLY EXPOSED. 176 MEN.

× LAGGERS 98 MEN.



1 = 0-9 2 = 10-19 3 = 20-29 4 = 30+ YEARS OF EXPOSURE

⊗ Dm = NOT ADJUSTED

Symptom and Sign Complexes in Radiographic Categories

The earlier analyses looked at men recorded as having a single symptom or sign; this analysis examines those men with combinations of symptoms and signs. Dyspnoea (grade 3+), rales, finger clubbing and rhonchi occur in the study population, and of these rales appear to be specifically associated with asbestosis. It was unusual for any of these changes to be present in isolation and it thus seems to be reasonable to group together these abnormal symptoms and signs in order to see whether or not the groupings are associated with patterns of change in lung function.

There are 16 different combinations. The numbers of men in each class are as follows: Numbers of Men with Normal X-rays

Group I	Number of Men
Rales	6
Rales and clubbing	0
Rales and dyspnoea	1
Rales, clubbing and dyspnoea	1
Clubbing	5
Clubbing and dyspnoea	1
Dyspnoea	7
Rales and rhonchi	3
Rales, rhonchi and clubbing	0
Rales, rhonchi, clubbing and dyspnoea	0
Rales, rhonchi and dyspnoea	0
Total	24

Group II	Number of Men
Rhonchi	11
Rhonchi and clubbing	0
Rhonchi and dyspnoea	1
Rhonchi, clubbing and dyspnoea	0
Total	12

Group III	
Men without abnormal symptoms or signs	144
Total number of men with normal radiographs in Groups I, II and III	180

Rales appear to be associated with pulmonary fibrosis; rhonchi appear to be related to airways obstruction. Rales and rhonchi seem to be the dominant signs which help to differentiate between these two broad patterns of lung disease. The combination of symptoms and signs have been allocated to the three groups shown above.

Group I contains men in whom the presence of rales dominates the picture. There are three men in this group who have rales and rhonchi, and they are included because the presence of rales may suggest pulmonary fibrosis. Men with clubbing, dyspnoea, or clubbing and dyspnoea are also included in this group because these changes are supposed to be associated with asbestosis.

Group II consists of men in whom the presence of rhonchi is dominant. Men who also have rales are excluded from this group.

Group III consists of men who have no abnormal symptoms or signs.

Table 4.XXXIV shows the numbers and percentages of men with combinations of symptoms and signs in x-ray categories. There are increasing proportions of men with rales, clubbing and dyspnoea, and combinations of these characteristics, with increasing radiographic abnormality. There is no such trend for men with rhonchi, clubbing and dyspnoea.

Lung Function Results of Men with Normal Radiographs

The lung function results for men in radiographic categories has been previously analysed (pages 167-8). In the following analysis the 180 men with normal radiographs (Category 0/0 without pleural abnormality) have been sub-divided into the three groups of symptom and sign complexes and mean values for lung function tests calculated for these groups. The age and height adjusted values are shown in figure 4.17. In comparison with the men without symptoms or signs the men with rhonchi, clubbing and dyspnoea have a decreased value for FVC, while those men with rales, clubbing and dyspnoea have an even lower value. The lowest value for $FEV_{1.0}$ and FEV% occurs in the group with rhonchi. Total lung capacity (TLC) is unaltered for men with rhonchi, but decreased in men with rales. Transfer factor (T_L) is unchanged in the men with rhonchi, but reduced for men with rales. The Transfer factor calculated using the "effective alveolar volume" (see calculation pages 254-67) shows a high value for the men thought to be normal, a lower value for those with rhonchi and an even lower value for men with rales.

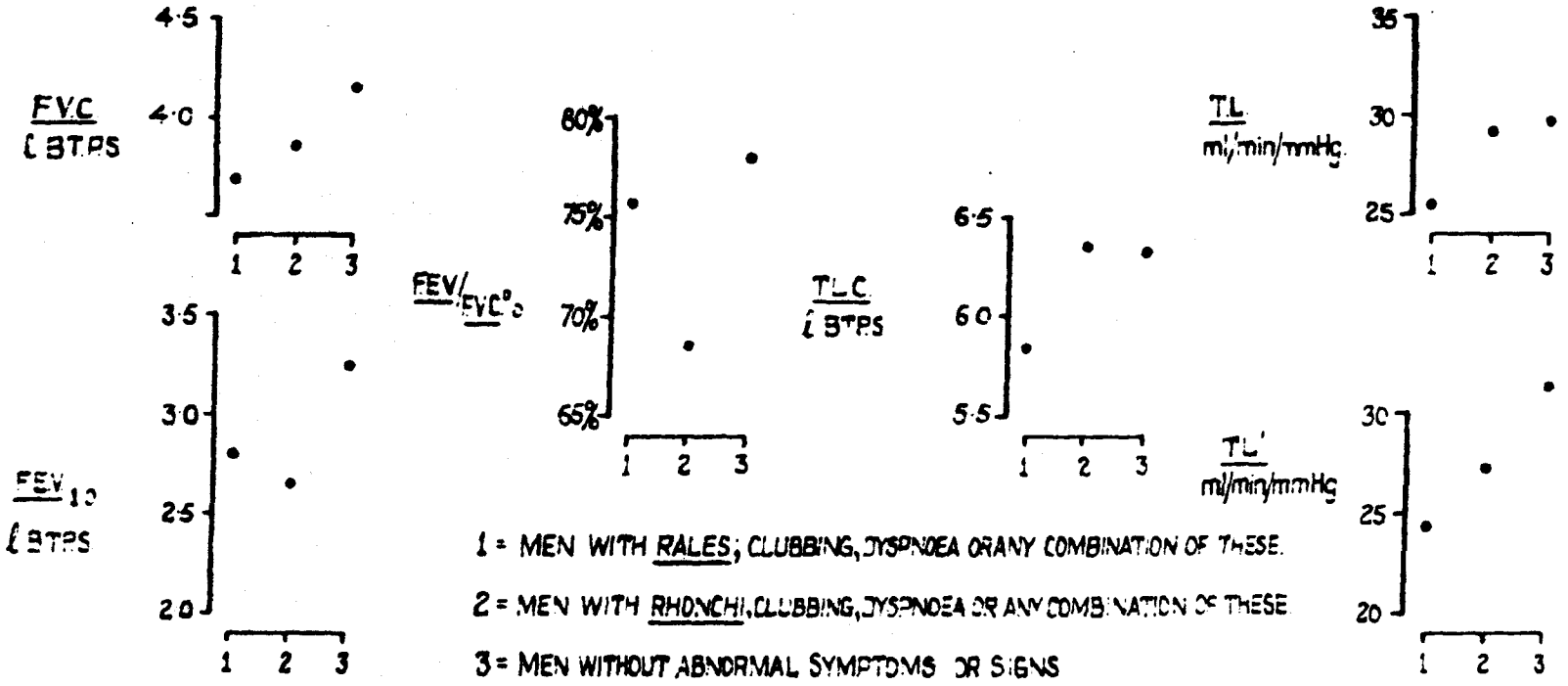
Hyponychial Angle

The mean values for the hyponychial angle are shown in figure 4.18 and in Tables 4.XV, 4.XXV and 4.XXVI.

FIG. 4.17.

*MEAN VALUES FOR LUNG FUNCTION TESTS FOR MEN WITH NORMAL X-RAY FILMS IN SYMPTOM AND SIGN COMPLEXES.

* ADJUSTED TO HT. 171 CMS. AGE 45 YEARS.



1 = MEN WITH RALES; CLUBBING, DYSPNOEA OR ANY COMBINATION OF THESE.

2 = MEN WITH RHONCHI, CLUBBING, DYSPNOEA OR ANY COMBINATION OF THESE

3 = MEN WITHOUT ABNORMAL SYMPTOMS OR SIGNS

The differences between occupational groups are small. The values are larger for the laggers and sprayers than for the other two groups, but the sprayers are older and an allowance has not been made for age. There appears to be some relationship with smoking categories and to x-ray abnormality. These differences in mean values of the angle cannot entirely be explained by differences in age.

The remaining results are briefly mentioned, but as so few men completed the tests it is probably not possible to draw reliable conclusions from them.

Standardized Ventilation

There were many men who could not complete the standardized exercise test for one reason or another. The mean values for those who did complete the test are shown in Tables 4.XIX-XXIII for occupational groups, and in Table 4.XXXV for x-ray abnormality. Figure 4.19 shows these values and it can be seen that the mean values are higher for laggers and sprayers than for the two groups with the least asbestos exposure. There is an increase in the mean value for standardized ventilation with increasing radiographic abnormality. There was no obvious relationship between weight and the standardized ventilation in this population.

Dyspnoea and Defects in Lung Function

There were even fewer men who completed the exercise ventilation, standardized ventilation and transfer factor estimations. The results for those who did complete all three tests were sub-divided into grades of dyspnoea and the mean values are shown in Table 4.XXXVI, figure 4.20. These data suggest that there is a relationship between dyspnoea category and values for exercise ventilation, standardized ventilation and transfer factor.

FIG. 4. 18.

HYPONYCHIAL ANGLE BY OCCUPATIONAL GROUP, SMOKING HABITS AND X-RAY CATEGORY.

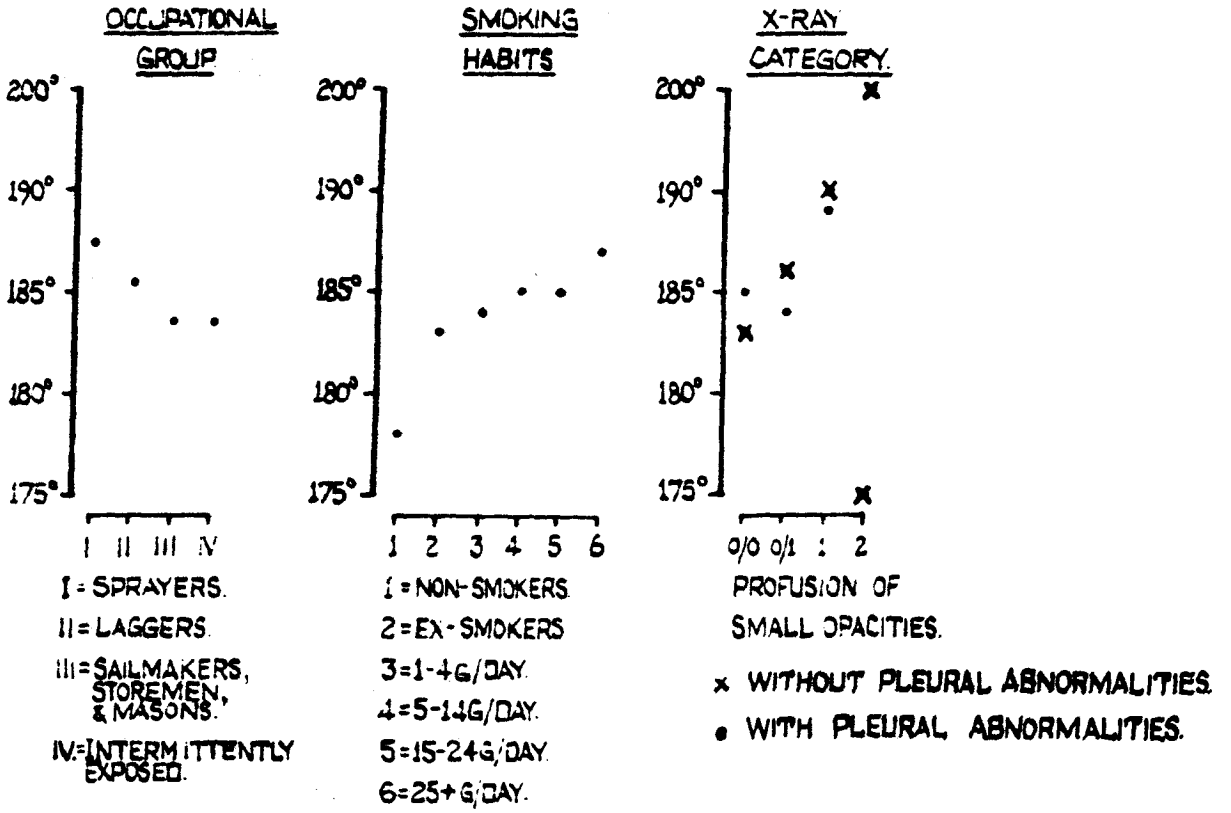


FIG. 4. 19

STANDARDIZED VENTILATION IN OCCUPATIONAL GROUPS, AND X-RAY ABNORMALITY.

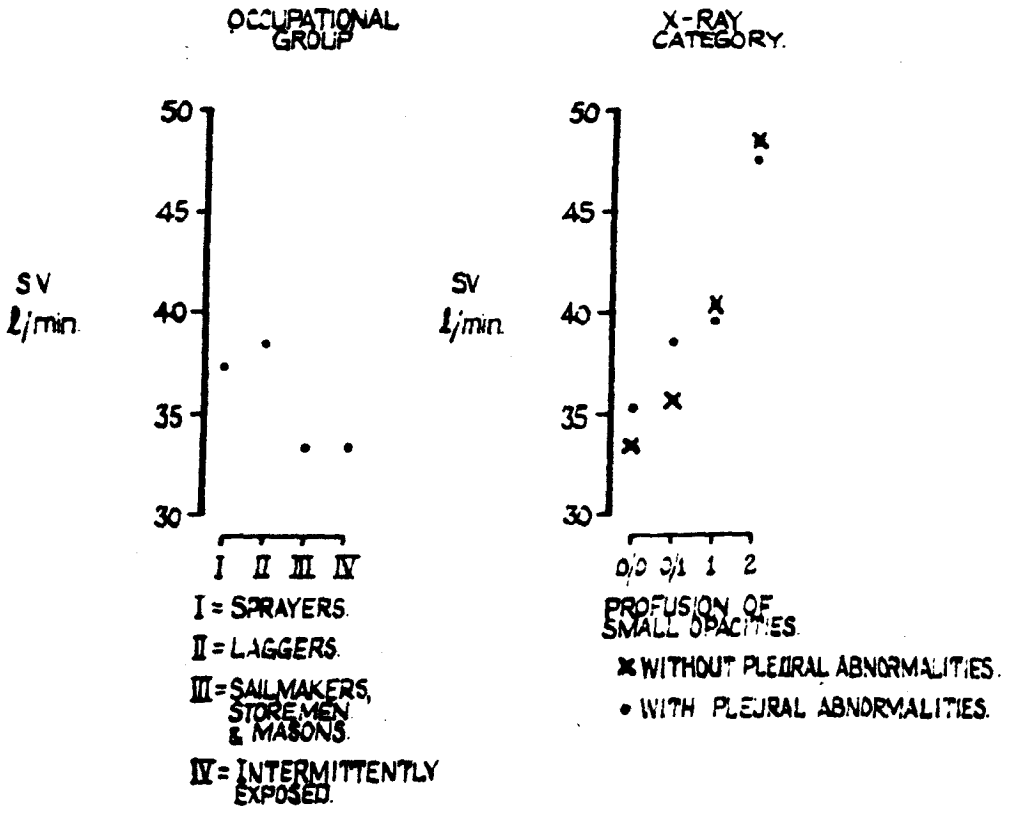
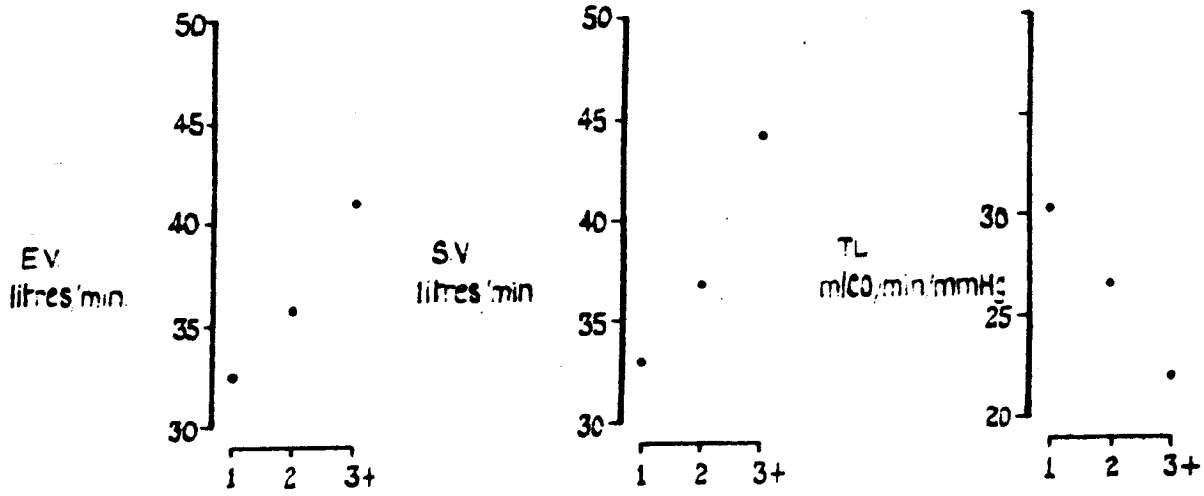


FIG. 4.20

EXERCISE VENTILATION; STANDARDIZED VENTILATION AND TRANSFER FACTOR IN GRADES OF DYSPNOEA



DYSPNOEA GRADE.

176 MEN WITH GRADE 1 DYSPNOEA.

80 MEN WITH GRADE 2 DYSPNOEA.

13 MEN WITH GRADE 3+ DYSPNOEA.

Discussion

The choice of occupational groups in this study was made after consideration of the dust concentrations, and the duration of exposure experienced by the men in each occupation. Spraying and stripping asbestos was associated with the most intense dust exposure. Stripping sprayed asbestos continues to occur from time to time, but spraying stopped in 1963, with relatively little of this work after 1958, and this explains why there are no sprayers in this study under the age of 35 years. The age distributions are not exactly the same in each group. Some allowance can be made for these differences and this will be explained later in this section.

The laggers have been exposed to high concentrations of asbestos dust when removing old pipe lagging, and to locally high concentrations when applying pipe and machinery insulation. The sailmakers, storemen and masons have not been exposed to such high dust concentrations as the laggers, but their work involved the manipulation of asbestos materials for most of their working time. They form an intermediate group between the laggers and the intermittently exposed group.

This last group, the intermittently exposed, is made up of men who have periodically worked alongside any of the other groups, and they have been haphazardly exposed to varying concentrations of asbestos dust.

It was hoped that by examining the men in these four groups, it might be possible to study the effects of intensity of exposure to asbestos as well as the effects of duration of exposure. Gross correlation between intensity and duration of exposure and the development of lung function and radiographic abnormality in Canadian asbestos miners was suggested by Wright (1955), but his paper did not give any supporting data. Poor correlation between

intensity and duration of exposure and lung function abnormality was reported by Bader et al (1961) in a study of 17 asbestos factory workers. They found that lung function changes correlated better with radiographic abnormality in their subjects. Kleinfeld et al (1966) also failed to find any correlation between duration of exposure and clinical, radiological or physiological abnormalities in a study of 56 asbestos workers.

There are many variable factors other than asbestos which might influence the findings in the population under consideration. Age, height, weight and smoking habits all have some effect on symptoms, signs, and lung function.

Age and Height

The lung function results have been adjusted to standardize for differences in age and height. This has not been done for weight because it does not appear to be so closely correlated with lung function.

If is difficult to apply similar standardization for symptoms and signs which might possibly be related to age. Three of the groups in this study are similar in mean age and height so that comparisons may be made between them. By considering years of exposure to asbestos we may make comparisons between all four groups.

Smoking Habits

There are small differences between the occupational groups in their smoking habits (figure 4.2, page 159). The effect of smoking on lung function (figure 4.7, page 169) shows that while the non-smokers have higher mean values for most tests of lung function than the ex-smokers or smokers, there is little

difference between the values in the smoking categories. The differences between the proportions of non-smokers in the occupational groups are small, so that although smoking has some effect on lung function it is reasonable to suggest that this effect will apply equally to each group.

Men excluded from the Analysis

The removal of any persons from a sample population may introduce bias, but it is unsatisfactory to include, especially for the analysis of lung function, men who have extensive pulmonary pathology due to other known conditions. It can be seen from Table 4.V (page 206) that the 22 cases excluded for this reason had defects which would have seriously altered their lung function results. It is possible that some of these men might possibly have been affected by asbestos exposure, but it is very difficult to make proper allowance for this.

Most of the eleven men exposed to dusts other than asbestos, and who are excluded for that reason from the analysis, had long exposure to asbestos (Table 4.VI, page 207). It is not possible to make allowance for the exposure to other dusts, so that it is better to exclude the men from the analysis.

The exclusion of the above 33 men should enable the relationship between exposure to asbestos and lung function to be more carefully considered.

Respiratory Symptoms and Signs

The results shown in figure 4.1 (page 157) suggest that there are more men with productive coughs for more than 3 months each year, and dyspnoea of grade 3 or more, in those groups most heavily exposed to asbestos. When the groups are considered for duration of exposure (figure 4.12, page 177) it can be seen that the sprayers and laggers have proportionately more men with

grade 3 dyspnoea for each 10 year period of exposure than do the remaining two groups.

There does not appear to be any obvious difference between the groups for those men with a recent history of chest illness (Question 12) or for men with a history of any kind of chest illness (Questions 22-27). This suggests that there has not been any self selection of men with previous chest diseases into the groups with heavy dust exposure. The converse should be true because such men should have been prevented from becoming laggers and sprayers at the medical examination required before starting these jobs.

Rales and clubbing (figure 4.3, page 161) appear to be related to the intensity, and duration of exposure to asbestos (figure 4.12, page 177), while rhonchi do not. Rales and clubbing, but not rhonchi, were described in cases of asbestosis amongst insulation and factory workers (Thomson et al 1965, Kleinfeld et al 1966, Leathart 1968), but those descriptions did not show any relationship between physical signs and intensity or duration of exposure.

The large proportions of men in each group with asbestos bodies or fibres in the sputum is not surprising in view of the earlier account of working methods and dust concentrations. These results help to confirm that many men not thought to have been exposed to asbestos have been exposed to an extent where they readily produce asbestos bodies or fibres in their sputum. The differences between occupational groups may partly be explained by the fact that more men in the heavily exposed groups had coughs and produced sputum than did the sailmakers, storemen, masons and intermittently exposed.

Asbestos corns were found in over 60% of the laggers and sailmakers, but in very few other men. Previous writers have suggested that the corns are due

to the penetration of the skin by the sharp, brittle amosite fibres (Alden and Howell 1944, Hunter 1969). In the present study this might be the explanation for the few cases occurring in the sprayers, storemen, masons and intermittently exposed. These men might have handled amosite fibre, or amosite lagging sections, or sheet asbestos and some of the fibre could have broken off in the skin of their hands.

This is not the explanation for the large proportion of sailmakers who have asbestos corns as their work involves the use of chrysotile asbestos cloth and the fine, silky fibres are not usually capable of penetrating the skin. The sailmakers, and laggers, use needles in their work and it is when they prick their fingers with the needles that they carry in small fibres of chrysotile asbestos. Most of the corns in the laggers are also due to needles pricking the skin, but some must also be due to handling materials containing amosite asbestos.

The corns appear within about two weeks unless the fibre is at once removed, and once present they remain until the last fragment of fibre has been removed. Most laggers state that they attempt to "dig out" fibre fragments as soon as they feel a corn developing.

Radiographic Abnormalities

The radiographic appearances of advanced cases of asbestosis have been well documented, and the radiographic changes accompanying "early" asbestosis, together with the pleural manifestations of the disease are now receiving more attention in the literature, (see Review of Literature, pages 10-40). An attempt has been made in the present survey to obtain an independent, objective reading of the radiographs by using the "combined reading score" of four readers (see page 144). There are inter- and intra- observer differences

in any study of this kind and the use of the combined readings attempts to minimize these differences.

It can be seen from the data (Tables 4.XVI, XVII and XXX, figures 4.4 and 4.13) that there is a relationship between radiographic abnormality and both the intensity and duration of exposure to asbestos. Table 4.XVIII (figure 4.5) shows that the radiographic abnormalities are related to those signs and symptoms associated with asbestosis, i.e. rales, clubbing and dyspnoea, but not to the presence of rhonchi. It is interesting to see in figure 4.5 that men in each radiographic category of small opacities with pleural abnormalities have more abnormal symptoms and signs than those without pleural abnormality. Radiographic abnormality is not associated with the occurrence of rhonchi.

There is increasing interest in the pleural abnormalities associated with asbestos exposure, and I consider the study of these manifestations of the disease to be very important in the future. In the present analysis the x-ray films were regarded as having pleural abnormality when one or more reader recorded a pleural abnormality which was not thought to be due to other conditions (e.g. tuberculosis, empyema, trauma). Most of these men with other chest pathology were previously excluded from the analysis (Table 4.V, page 206).

The pleural abnormalities which were recorded included hyaline pleural plaques, calcified pleural plaques and diffuse pleural thickening. The majority of positive readings were noted only by a single reader and were recorded as hyaline pleural thickening. There were only three cases in which extensive, diffuse pleural thickening was present, and only 11 cases where pleural calcification was recorded. The pleural lesions therefore represent minimal pleural abnormalities, and it is of interest to see what effect these abnormalities seem to have produced in the way of symptoms, signs and lung function changes.

Lung Function Tests

Most of the published reports of pulmonary function tests in persons exposed to asbestos have described the findings in rather small groups of men, and in several of these reports subjects diagnosed as having asbestosis were compared with men not so diagnosed, or with men with no known exposure to asbestos (see Review of Literature pages 10-40). Men with asbestosis were shown in these reports to have a restrictive ventilatory defect with reduced lung compliance and low transfer factor. Hunt (1965) suggested that there were relationships between intensity and duration of exposure and lung function results, but Bader et al (1961) and Kleinfeld et al (1966) failed to demonstrate any such relationship.

Lung Function and Intensity of Exposure

The results in the present survey show that there are relationships between the intensity of exposure and the lung function defects associated with asbestosis (figure 4.6, page 168). The use of an independent assessment of the lung function categories (see page 171) helps to confirm the association between intensity of exposure and the development of lung function abnormalities (figure 4.9, page 173). These assessments take into account the age, height and weight of each of the subjects, but nothing else, so that they are a valuable method of comparing the occupational groups. It can be seen that there are consistent trends for those defects associated with asbestosis to occur more frequently in the most highly exposed groups.

Lung Function and Duration of Exposure

There are not such clear relationships between duration of exposure and lung function results (figures 4.15 and 4.16, pages 181-2). The lung

function assessments (figure 4.14, page 179) suggest that there are fewer men with normal assessments for each 10 year period of exposure in the most highly exposed groups. This is a reflection of the intensity of exposure. The results of these assessments for the occupational groups, excluding the sprayers, do show a trend for fewer normal assessments to occur in each group as the duration of exposure increases. Figure 4.16 (page 182) shows the comparison between the laggers and intermittently exposed, and it can be seen that in the former the results of all the tests, excluding residual volume (RV), fall off with increasing duration of exposure.

Lung Function Results and Radiographic Abnormalities

The classification of men according to the degree of x-ray abnormality associated with asbestos exposure should also sub-divide them into degrees of progressive lung function defects, if those defects are also associated with asbestos exposure. The data from the present survey indicate that this is so (figure 4.8, page 170). The results also show that men with pleural abnormalities have lower values for the lung function tests than those without. The pleural changes are minimal and the differences between lung function values are small, but the trends are consistent.

Table XXXVII (page 238) shows the data on eleven men in this population who had calcified pleural plaques. The age adjusted values for this group are just lower than for men without small opacities but with pleural abnormality (figure 4.8, page 170). The numbers of men in the different categories of small opacities who have pleural calcification are too small to draw valid conclusions, but it would appear that these results are in accord

with the observation by Leathart (1968c) that pleural calcification alone is not accompanied by lung function defects. In another communication Leathart (1968b) suggested that hyaline pleural plaques were not associated with lung function abnormalities, but Becklake et al (1968) presented data which suggests that pleural abnormality is accompanied by small defects in lung function.

It has previously been shown in the present survey that men with pleural abnormalities have proportionately more symptoms and signs associated with asbestosis (figure 4.5, page 165) than men without these changes, so that it should not be surprising to find that men with pleural abnormalities also have the defects in lung function associated with asbestosis.

Airways Obstructive Disease

It is clear from the data that signs and lung function changes associated with airways obstructive disease are not associated with the disease process occurring in the men in this study. Rhonchi were not associated with intensity of exposure (figure 4.3, page 161), duration of exposure (figure 4.12, page 177), or radiographic abnormality (figure 4.5, page 165). The age and height adjusted mean values for the FEV% in the four occupational groups are 75.7% for sprayers, 75.8% laggers, 76.1% sailmakers, and 76.7% intermittently exposed. These differences are not significant, but there is a significant trend in the decreasing value of FVC and $FEV_{1.0}$ with occupational group. The same tendency is seen for lung function in x-ray category where the $FEV_{1.0}$ value is reduced proportionately with the value for FVC. Figure 4.17 (page 186) shows quite clearly that, for men with normal films, those who have rhonchi have the lowest value for the FEV%, but none of the defects associated with pulmonary fibrosis. The reduced value for T_L1 (Transfer factor calculated using the effective alveolar volume) is a reflection of some

inequality in ventilation and perfusion in the group with rhonchi.

Hyponychial Angle

The value of this measurement has not clearly emerged from this study. Thirty-seven men have early or definite finger clubbing. In three the angle is less than 184°; in 21 between 185°-194°; in 10 men between 195°-204°; and 3 men have an angle of more than 205°. Regan et al (1967) gave 208° as the mean value for the seven men in their study thought to have clubbed fingers, and 197° as the mean value for 25 men with doubtfully clubbed fingers. The mean angle for the 18 of their subjects considered normal was 187°.

The measurement of this angle does not take into account the other factors considered by the physician in his clinical assessment of finger clubbing. These factors include the convexities of the nail, the fluctuation at the nail bed, and the area of erythema at the nail bed.

In general there is agreement between the assessment of the degree of finger clubbing and the size of the hyponychial angle, but the values so obtained appear to be lower than those reported by Regan et al. The data from the present survey suggest relationships between the magnitude of the hyponychial angle and the intensity of exposure to asbestos, and to radiological abnormality but these differences may be largely explained by differences in age of the groups under consideration. A more detailed study is required to assess the true value of this measurement.

The diagnosis of Asbestosis

The difficulties of confirming the diagnosis of asbestosis have been mentioned by Leathart (1968), (Mackenzie and Harries 1969). Leathart points out that the Pneumoconiosis Medical Boards have had more experience in the diagnosis of asbestosis than anyone in Britain, and this is the reason why

many British reports compare cases certified by the Medical Boards with men who do not have the disease. There are pitfalls in these comparisons, the main one being that we are not usually told whether or not the Medical Boards also examined the "control" groups. If they did not then we do not know if some of those men might have the disease.

The disease process is a progressive pulmonary fibrosis associated with asbestos exposure, and the criteria (used by the Medical Boards) for making the diagnosis of asbestosis have been described in a Ministry of Social Security booklet (1967). This booklet states that the diagnosis of asbestosis should be considered when there is evidence of exposure to asbestos and two or more of the following criteria are present; dyspnoea; basal rales; finger clubbing; radiographic evidence suggesting pulmonary fibrosis; reduced transfer factor. There is little guidance as to what constitutes "adequate exposure" to asbestos in order to produce the disease, but McVittie (1965), for many years a senior member of one Medical Board, suggests that in some occupations 3-4 years of recent exposure is adequate, but in general about 8 years exposure is required. He points out that very short exposure might prove sufficient if the exposure took place many years previously.

The results from the present study show that defects conforming to these criteria are occurring in the survey population, and are associated with the intensity and duration of exposure. Thirty-six men from the study population have been accepted by the Pneumoconiosis Medical Panel as suffering from asbestosis. The data relating to these men are shown in Table 4.XXXVIII (page 239). It can be seen that most of the men were in the highly exposed groups, and the mean duration of exposure was 22.3 years. Rales were found in 32 of the 36, but rhonchi in only 3 of them. The lung function results

show that they have a restrictive defect, with no airways obstruction and reduced transfer factor. Only one man was thought to have a completely normal chest radiograph, and pleural abnormalities were recorded in 28 of the 36 men.

There have been conflicting views on the value of physical signs, especially basal rales, in the diagnosis of asbestosis, but it is now accepted that these signs are present in almost all established cases of asbestosis. Leathart (1968) says that they are present in nearly all established cases of asbestosis, and early in the disease they may be heard at one examination but not on subsequent occasions. This has also been my experience, and I consider the presence of the typical, dry, crackling rales heard at the lung bases, anteriorly and in the axillae at first, and later in the disease over more and more of the lung fields, to be of great diagnostic importance.

I consider that exposure to asbestos sufficient to cause pleural calcification, or pleural fibrosis is most likely to have also caused some interstitial pulmonary fibrosis, even though this might be microscopical in extent. I agree that in the majority of these cases there is no evidence of clinical disability or lung function abnormality, but there are those cases in which there is extensive, constrictive pleural fibrosis or pleural calcification which does cause considerable disability. As all these manifestations are part of the same disease process, I consider that it would be more realistic to widen the definition of "asbestosis" as laid down in the

National Insurance (Industrial Injuries) Act (1948), to include those cases in which the pleural manifestations cause disability.

"Early Diagnosis"

There is evidence from this survey that when basal rales occur in the men with "normal" radiographs they are accompanied by the lung function defects associated with asbestosis (figure 4.17, page 186). This suggests to me that clinical examination of men exposed to asbestos continues to be an equally important part of their medical supervision as radiological or physiological examination. Williams and Hugh Jones (1960) and Hunt (1965) suggested that reduction in transfer factor might precede radiological evidence of asbestosis. It may be that the men with clinical and physiological defects occurring in the group with normal x-rays in the present survey will proceed to develop more evidence of the disease, but that is all that can be said at present.

There is nothing in the data presented from this survey which suggests that any one form of examination, or test, is superior to all the others in making the diagnosis of asbestosis. Careful consideration of the history of exposure to asbestos, the presence of symptoms and signs, especially dyspnoea, basal rales, and finger clubbing, together with radiographic examination of the chest and assessment of the results of lung function tests, are required before an attempt can be made to make the diagnosis. Other causes of pulmonary fibrosis should be excluded.

The tests of lung function found to be most useful are the tests of ventilatory capacity, the sub-divisions of lung volume, and the estimation of transfer factor. Not enough men completed the standard exercise test to draw firm conclusions of its value, but it is suggested that this test may prove to

be valuable. More work is required to gain more experience using an improved method of estimating the ventilatory cost of exercise.

There does appear to be correlation between the grade of dyspnoea and the values for exercise ventilation, standardized ventilation and transfer factor. As this might be important in the objective assessment of disability it is obviously desirable that more work should be undertaken to try to clarify the situation.

Further analyses are being undertaken, elsewhere, to attempt to relate estimates of exposure to physiology, radiology, signs and symptoms, and to find optional ways of combining evidence to support a diagnosis of asbestos disease.

Summary

Three hundred and sixty-nine men exposed to asbestos for different lengths of time, and different intensities of dust concentration were examined clinically, radiologically and physiologically. Independent assessments of the radiographs were made by a panel of experts. Lung function results were also independently assessed by a leading respiratory physiologist.

There is evidence of relationships existing between those defects associated with asbestosis and both the intensity and duration of exposure to asbestos. Men with pleural abnormality in addition to the appearance of small opacities on their chest radiographs were shown to have more symptoms and signs, and lower lung function values, than men without pleural abnormality.

Productive cough and dyspnoea grade 3 or more, basal rales and finger clubbing were the most important symptoms and signs associated with the intensity and duration of exposure, and with the radiographic abnormality. The tests of ventilatory capacity, lung volumes and transfer factor were

associated with the intensity of exposure and had also some relationship to the duration of exposure.

Men with normal radiographs who had rales, clubbing and dyspnoea, or combinations of these signs and symptoms showed a restrictive ventilatory defect with a loss of transfer factor suggesting that these signs and symptoms might be evidence of pulmonary fibrosis occurring in men without detected radiological abnormality.

The ventilatory cost of exercise as measured by the standardized ventilation showed some relationship to the intensity of exposure and x-ray abnormality, but insufficient men completed the test to draw valid conclusions from the results.

TABLE 4.V

Men excluded from analysis of clinical and physiological tests

Group	Number of Men	Cause for Exclusion
Sprayers	2	2. Extensive healed pulmonary tuberculosis.
Laggers	2	1. Pulmonary tuberculosis. 1. Old empyema with extensive pleural fibrosis.
Sailmakers	2	1. Severe kyphosioliosis. 1. Sarcoidosis.
Storemen and Masons	8	2. Thoracotomy. 1. Thoracoplasty. 1. Lobectomy. 1. Carcinoma of lung + radiation fibrosis. 1. Old empyema. 1. Severe bony chest abnormality. 1. Very low I.Q. Incapable of performing even the simplest test.
Intermittently exposed	8	3. Extensive healed pulmonary tuberculosis. 2. Pulmonary tuberculosis. 1. Thyrotoxicosis. 1. Carcinoma of lung. 1. Severe bony chest abnormality.
TOTAL	22 Men	

TABLE 4.VI

Eleven Men with Exposure to Dusts other than Asbestos

Dockyard Occupational Group		Asbestos Exposure Years	Other Dust Exposure	
			Type	Duration Years
Laggers	(1)	3	Quarrying	5
	(2)	13	Quarrying	3
	(3)	28	Quarrying	5
	(4)	17	HM Dockyard Foundry	6
	(5)	16	HM Dockyard Foundry	5
	(6)	16	Coal Miner	5
	(7)	9	China Clay Worker	11
Intermittent Exposure	(8)	10	HM Dockyard Foundry	3
Storeman	(9)	44	Boiler Scaler	1
Mason	(10)	18	Coal Miner	4
Mason	(11)	3	Gold Miner	3

( ) Numbers in brackets refer to the columns of data for each man shown in the appendix to this section (pages).

TABLE 4.VII

Numbers of men in occupational groups included in the analyses of data

Sprayers	50
Laggers	98
Sailmakers, Storemen and Masons	45
Intermittently exposed	176
TOTAL	369

TABLE 4.VIII

Age Analysis. Number of men in 5 year age groups.

Ages	Number of Men				
	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
15-19	3	0	1	1	1
20-24	23	0	6	4	13
25-29	38	0	14	7	17
30-34	38	0	10	3	25
35-39	29	2	10	1	16
40-44	42	10	13	3	16
45-49	52	6	15	5	26
50-54	46	9	7	9	21
55-59	40	8	9	10	13
60-64	46	9	12	1	24
65-69	11	5	1	1	4
70-74	1	1	0	0	0
TOTAL	369	50	98	45	176

Age in Years

Age in Years	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
Mean Years	44.56	53.36	42.52	43.82	43.39
Standard Deviation	13.07	8.77	12.76	13.51	13.26
Maximum Years	70	70	65	65	69
Minimum Years	18	38	19	18	19

TABLE 4.IX

Time from first exposure: Number of men in 5 year periods from first exposure

Years from first exposure	Number of Men				
	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
0- 4	58	0	31	11	16
5- 9	70	0	24	18	28
10-14	49	1	7	3	38
15-19	52	14	9	5	24
20-24	60	20	14	5	21
25-29	33	7	4	1	21
30-34	27	6	4	1	16
35-39	12	0	2	1	9
40-44	4	0	2	0	2
45-49	4	2	1	0	1
	369	50	98	45	176
		Years from first exposure			
Mean Years	16.11	22.82	12.03	10.62	17.89
Standard Deviation	10.72	7.04	11.02	8.89	10.36
Maximum Years	47	47	45	37	47
Minimum Years	1	11	1	1	2

TABLE 4.X

Duration of exposure: Number of men in 5 year periods of exposure

Years of Exposure	Number of Men				
	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
0- 4	70	4	33	12	21
5- 9	73	0	22	19	32
10-14	54	6	8	3	37
15-19	52	16	8	4	24
20-24	55	12	18	4	21
25-29	30	9	4	1	16
30-34	19	1	1	2	15
35-39	13	1	4	0	8
40-45	1	0	0	0	1
45-49	2	1	0	0	1
TOTAL	369	50	98	45	176

Duration of Exposure in Years

Years of Exposure	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
Mean Years	14.75	19.46	11.24	9.84	16.63
Standard Deviation	10.17	8.36	9.76	8.44	10.22
Maximum Years	47	47	36	32	47
Minimum Years	1	1	1	1	1

TABLE 4.XI

Height of the Subjects in Centimetres

Height cms.	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
Mean Ht.	170.6	169.5	171.1	171.6	170.4
Standard Deviation	7.1	5.6	7.6	6.4	7.3
Maximum Ht.	198.0	185.0	192.0	185.0	198.0
Minimum Ht.	135.0	154.0	150.0	152.0	135.0

TABLE 4.XII

Respiratory Symptoms and Previous Chest Illness in Occupational Groups

Occupational Group	Sprayers		Laggers		Sailmakers, Storemen & Masons		Intermittently exposed	
No. in Group	50		98		45		176	
	No.	%	No.	%	No.	%	No.	%
Cough & Phlegm	18	36	27	28	3	6	33	19
Increased Cough for 3 Weeks	3	6	1	1	3	6	4	2
Breathlessness								
Grade 0	2	4	3	3	1	2	1	1
1	10	20	52	53	29	65	120	68
2	25	50	29	30	14	31	44	25
3	12	24	13	13	1	2	7	4
4	0	0	0	0	0	0	0	0
5	1	2	1	1	0	0	4	2
Chest Illness in last 3 years	10	20	8	8	7	15	23	13
Previous Chest Illness	24	48	34	35	14	30	84	48
Heart Disease	3	6	4	4	0	0	3	2

TABLE 4.XIII

Smoking Habits in Occupational Group

Group	Sprayers		Laggers		Sailmakers, Storemen & Masons		Intermittently Exposed		All Groups	
	No.	%	No.	%	No.	%	No.	%	No.	%
Non-smoker	7	14	8	8	8	18	24	14	47	13
Ex-smoker	7	14	24	24	10	22	29	17	70	19
1-4G	2	4	4	4	3	7	9	5	18	5
5-14G	13	26	27	28	4	9	36	20	80	22
15-24G	16	32	23	23	11	24	58	33	108	29
25+G	5	10	12	12	9	20	20	11	46	12
TOTALS	50	100%	98	100%	45	100%	176	100%	369	100%

TABLE 4.XIV

Physical Signs in Occupational Groups

Occupational Group	Sprayers		Laggers		Sailmakers, Storemen & Masons		Intermittently exposed	
No. in Group	50		98		45		176	
	No.	%	No.	%	No.	%	No.	%
Rales	29	58	18	18	8	18	17	10
Rhonchi	8	16	16	16	3	7	16	9
Finger Clubbing	15	30	9	9	1	2	12	7
Asbestos bodies or fibres in sputum	28	56	59	60	16	36	77	44
Asbestos Corns	2	4	61	62	9	20	4	2

*13 Sailmakers 8 or 62% had asbestos corns.

34 Masons and Storemen 1 or 3% had asbestos corns.

TABLE 4.XV

Mean Hyponychial Angle in Occupational Groups

Hyponychial Angle	All Groups	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
Mean Angle°	184.5°	187.5°	185.5°	183.4°	183.4°
Standard Deviation	7.3	8.8	7.4	7.2	6.5
Maximum Angle	227°	227°	208°	204°	202°
Minimum Angle	166°	173°	172°	171°	166°

TABLE 4.XVI

X-ray Classification in Occupational Groups

X-ray Category		Group	Sprayers	Laggers	Sailmakers, Storemen & Masons	Intermittently exposed
Profusion of Nodules	Pleural Abnormality	TOTALS	50	98	45	176
0	Absent	180	14	48	29	89
0	Present	61	8	15	3	35
0/1	Absent	55	8	15	4	28
0/1	Present	42	8	12	3	19
1	Absent	12	4	3	3	2
1	Present	14	5	4	2	3
2	Absent	1	0	0	1	0
2	Present	4	3	1	0	0

TABLE 4.XVII

Mean Years of Asbestos Exposure in X-ray Categories

Profusion of Opacities	0/0		0/1		1		2	
Pleural Abnormality	-	+	-	+	-	+	-	+
No. of Men	180	61	55	42	12	14	1	4
Mean Age Years	38.7	48.0	48.6	50.9	55.9	58.1	30.0	56.3
Mean Height cms.	171.1	168.1	171.6	170.7	170.6	170.4	172.0	175.0
Mean Years of Exposure	10.5	17.9	16.7	20.3	20.6	23.8	6.0	23.8
Mean Years since First Exposure	11.6	19.8	18.2	21.0	22.8	25.6	6.0	24.5

TABLE 4.XVIII

Numbers of Men with Physical Signs and Dyspnoea Grade 3+ in X-ray Categories

X-ray Category		Numbers in X-ray Category	Numbers of Men with Signs			
Profusion of Nodules	Pleural Abnormality		Rales	Rhonchi	Dyspnoea	Clubbing
0/0	Absent	180	11	15	11	7
0/0	Present	61	13	8	6	8
0/1	Absent	55	13	9	5	3
0/1	Present	42	19	7	8	8
1	Absent	12	4	2	2	5
1	Present	14	9	2	4	3
2	Absent	1	0	0	0	0
2	Present	4	3	0	3	3
TOTALS		369	72	43	39	37

TABLE 4.XIX

Mean Values, Standard Deviation and Regression on Age for Total Population

Test	No. Tested	Mean Value	Standard Deviation	Maximum Value	Minimum Value	Regression on Age	
						Slope	Constant
FVC	369	3.91	0.88	6.39	1.64	-0.047	6.041
FEV	369	3.00	0.84	5.56	0.67	-0.047	5.130
FEV/FVC%	369	75.85	9.88	97.30	28.59	-0.299	89.216
EV	286	34.05	6.61	58.40	13.29	0.154	27.670
SV	282	35.11	7.88	75.19	20.60	0.258	24.295
SVC	366	4.19	0.91	6.90	1.49	-0.046	6.292
RV	366	1.77	0.61	4.38	0.61	0.020	0.850
RV (10 sec.)	367	1.48	0.36	3.22	0.69	0.010	1.001
$\frac{\text{RV 10 sec.}}{\text{RV}}$	366	0.87	0.20	1.66	0.45	-0.003	1.051
TLC	366	5.97	0.96	8.82	3.01	-0.026	7.142
RV/TLC%	366	29.90	9.20	63.60	12.00	0.470	8.680
T_L	366	27.45	6.95	48.30	8.60	-0.297	40.740
T_L1	367	26.04	6.97	45.80	6.60	-0.337	41.110
D_M	364	42.75	11.38	79.10	11.20	-0.418	61.397

Lung Volumes expressed in litres BTPS. EV and SV in litres/min. T_L, T_L1 and D_M in mlCO/min/mmHg.

TABLE 4.XX

Mean Values, Standard Deviation and Regression on Age for Sprayers

Test	No. Tested	Mean Value	Standard Deviation	Maximum Value	Minimum Value	Regression on Age	
						Slope	Constant
FVC	50	3.43	0.73	5.12	1.74	-0.051	6.168
FEV	50	2.46	0.65	3.88	1.25	-0.044	4.827
FEV/FVC%	50	71.74	9.69	87.50	40.00	-0.211	83.051
EV	31	35.33	6.19	53.19	27.09	-0.072	38.839
SV	33	37.09	7.09	60.80	28.59	-0.016	37.885
SVC	50	3.67	0.74	5.34	1.95	-0.049	6.326
RV	50	1.96	0.61	4.34	0.97	0.020	0.880
RV (10 sec.)	50	1.62	0.45	3.22	0.90	0.011	1.040
$\frac{\text{RV 10 sec.}}{\text{RV}}$	50	0.86	0.22	1.56	0.45	-0.001	0.951
TLC	50	5.63	0.88	7.35	3.01	-0.029	7.201
RV/TLC%	50	34.70	8.70	62.30	21.20	0.540	5.690
T_L	50	22.17	6.13	40.90	8.80	-0.325	39.522
$T_L{}^1$	50	20.91	6.17	38.50	6.60	-0.338	38.959
D_M	49	35.57	10.17	62.30	11.20	-0.433	58.669

Lung Volumes expressed in litres BTPS. EV and SV in litres/min. T_L, $T_L{}^1$ and D_M in mlCO/min/mmHg.

TABLE 4.XXI

Mean Values, Standard Deviation and Regression on Age for Laggers

Test	No. Tested	Mean Value	Standard Deviation	Maximum Value	Minimum Value	Regression on Age	
						Slope	Constant
FVC	98	3.93	0.83	6.27	1.82	-0.046	5.901
FEV	98	3.06	0.75	4.94	1.39	-0.044	4.970
FEV/FVC%	98	77.58	6.99	97.30	59.09	-0.217	86.822
EV	78	36.07	7.69	58.40	21.00	0.211	27.477
SV	80	38.16	10.36	75.19	22.20	0.442	19.691
SVC	97	4.08	0.86	6.15	1.95	-0.045	6.041
RV	97	1.55	0.51	3.80	0.61	0.017	0.807
RV (10 sec.)	97	1.42	0.32	2.18	0.85	0.006	1.144
$\frac{\text{RV 10 sec.}}{\text{RV}}$	97	0.97	0.23	1.66	0.45	-0.005	1.227
TLC	97	5.63	0.86	8.04	3.84	-0.028	6.847
RV/TLC%	97	27.70	8.90	60.00	12.00	0.440	8.520
T_L	97	26.13	6.09	44.09	9.60	-0.263	37.400
T_L1	97	25.56	6.49	45.80	9.50	-0.311	38.881
D_M	97	40.81	10.26	71.80	11.50	-0.425	59.001

Lung Volumes expressed in litres BTPS. EV and SV in litres/min. T_L, T_L1 and D_M in mlCO/min/mmHg.

TABLE 4.XXII

Mean Values, Standard Deviation and Regression on Age for Sailmakers, Storemen and Masons

Test	No. Tested	Mean Value	Standard Deviation	Maximum Value	Minimum Value	Regression on Age	
						Slope	Constant
FVC	45	3.93	0.87	6.22	2.17	-0.037	5.585
FEV	45	3.02	0.86	4.57	1.16	-0.049	5.176
FEV/FVC%	45	76.16	11.41	90.19	46.20	-0.512	98.642
EV	37	33.56	6.70	49.90	23.29	0.097	29.429
SV	34	33.31	6.46	48.40	24.60	0.122	28.099
SVC	45	4.27	0.92	6.90	2.66	-0.033	5.742
RV	45	1.83	0.67	3.41	0.94	0.032	0.427
RV (10 sec.)	45	1.51	0.41	2.44	0.75	0.015	0.816
$\frac{\text{RV 10 sec.}}{\text{RV}}$	45	0.86	0.18	1.37	0.45	-0.005	1.126
TLC	45	6.10	1.07	8.82	3.60	-0.001	6.173
RV/TLC%	45	29.90	9.00	55.60	16.70	0.510	7.330
T_L	45	28.58	6.39	44.59	15.70	-0.259	39.971
T_L1	45	27.15	6.77	40.40	11.80	-0.323	41.306
D_M	45	43.33	9.96	76.30	26.59	-0.320	57.366

Lung Volumes expressed in litres BTPS. EV and SV in litres/min. T_L, T_L1 and D_M in mlCO/min/mmHg.

TABLE 4.XXIII

Mean Values, Standard Deviation and Regression on Age for Intermittent Exposure

Test	No. Tested	Mean Value	Standard Deviation	Maximum Value	Minimum Value	Regression on Age	
						Slope	Constant
FVC	176	4.04	0.90	6.39	1.64	-0.049	6.196
FEV	176	3.10	0.89	5.56	0.67	-0.047	5.180
FEV/FVC%	176	75.96	10.61	95.40	28.59	-0.270	87.717
EV	140	32.78	5.71	55.09	13.29	0.150	26.732
SV	135	33.27	5.83	53.69	20.60	0.190	25.632
SVC	174	4.39	0.92	6.49	1.49	-0.049	6.528
RV	174	1.83	0.61	4.38	0.73	0.019	1.010
RV (10 sec.)	175	1.46	0.34	2.55	0.69	0.010	1.005
$\frac{\text{RV 10 sec.}}{\text{RV}}$	174	0.83	0.17	1.28	0.45	-0.002	0.948
TLC	174	6.23	0.93	8.62	3.39	-0.030	7.537
RV/TLC%	174	29.70	9.20	63.60	12.20	0.450	9.980
T_L	174	29.42	6.86	48.30	8.60	-0.281	41.649
$T_L{}^1$	175	27.49	6.82	44.30	7.60	-0.321	41.471
D_M	173	45.72	11.59	79.10	12.50	-0.386	62.455

Lung Volumes expressed in litres BTPS. EV and SV in litres/min. T_L, $T_L{}^1$ and D_M in mlCO/min/mmHg.

TABLE 4.XXIV

Regression Coefficients for Lung Function Tests in Normal Males (Cotes 1965)

Index	Units	Constant term	Regression Coefficients	
			Height (cms)	Age (years)
FVC	litres	- 3.60	0.052	-0.022
$FEV_{1.0}$	litres	- 1.41	0.036	-0.031
RV	litres	- 3.45	0.027	0.017
TLC	litres	- 8.49	0.087	
T_L	mlCO/min/mmHg	-17.60	0.325	-0.220

TABLE 4.XXV

Mean Values for Lung Function Tests in Smoking Categories

Smoking Category	Non-smokers	Ex-smokers	1-4g	5-14g	15-24g	25g+
Number of Men	47	70	18	80	108	46
FVC	4.29	3.72	3.94	3.78	4.05	3.78
$FEV_{1.0}$	3.50	2.86	3.07	2.87	3.05	2.78
FEV/FVC%	82%	77%	78%	76%	75%	74%
SVC	4.58 *	4.08	4.24	3.95	4.33	4.10
RV	1.61 *	1.72 *	1.62	1.89 *	1.82	1.84
TLC	6.18 *	5.80 *	5.86	5.83 *	6.14	5.94
RV/TLC%	26% *	30% *	28%	32% *	30%	31%
$T_L CO$	33.0 *	29.0 *	27.5	25.4 *	26.5	25.3
$T_L 1$	31.6 *	27.3	26.7	23.9 *	25.2	23.9
D_M	49.8 *	43.7 *	43.9	39.9 **	41.8 *	41.2
Hyponychial Angle°	178 *	183	184	185 *	185 **	187 *
Years of Exposure	13.2	17.2	10.9	15.0	13.9	15.4
Age (years)	39.5	48.7	40.4	46.5	43.1	45.2
Height (cms)	170.3	171.1	169.8	169.6	171.1	171.1
Weight (Kg)	73.6	76.8	71.7	69.4	73.6	76.0

* One man in this group did not perform this test.

** Two men in this group did not perform this test.

Lung Volumes expressed in litres BTPS.

T_L, $T_L 1$, D_M in mlCO/min/mmHg.

TABLE 4.XXVI

Mean Values for Lung Function Tests in X-ray Categories

Profusion of Opacities	0/0		0/1		1		2	
Pleural Abnormality	-	+	-	+	-	+	-	+
Number of Men	180	61	55	42	12	14	1	4
FVC	4.23	3.71	3.86	3.50	3.42	2.93	4.89	3.14
$FEV_{1.0}$	3.31	2.75	2.96	2.55	2.49	2.16	4.21	2.12
RV	1.68*	1.84*	1.87	1.91*	2.08	1.71	1.93	2.17
TLC	6.19*	5.89*	6.00	5.59*	5.74	4.94	7.23	5.62
T_L	30.2*	25.4*	26.0	24.7*	23.2	21.1	44.6	16.8
T_L1	29.0*	23.5	24.6	23.2*	21.0	20.0	39.9	15.7
D_M	46.3**	38.8	43.1	37.6	37.3	35.8	76.3	31.6
Hyponychial Angle	183	185	186	184	190	189	175	199
Age	38.7	48.0	48.6	50.9	55.9	58.1	30.0	56.3
Height	171.0	168.1	171.6	170.7	170.6	170.4	172.0	175.0

Lung Volumes expressed in litres BTPS.

T_L, T_L1, D_M in mlCO/min/mmHg.

* One man in this group did not perform this test

** Three men in this group did not perform this test

TABLE 4.XXVII

Lung Function Assessment in Occupational Groups

Lung Function Category	Sprayers		Laggers		Sailmakers, Storemen & Masons		Intermittently exposed	
	No.	%	No.	%	No.	%	No.	%
Normal	23	46	61	62	34	75	145	82
Transfer Defect	9	18	11	11	1	2	4	2
Restricted TLC	7	14	17	18	3	7	7	4
Transfer Defect + Restricted TLC	5	10	9	9	2	4	0	0
Obstructive Defect	2	4	0	0	1	2	8	5
Other Defects	1	2	0	0	2	4	2	1
Doubtful Transfer or Restricted Defects	0	0	0	0	2	4	5	3
Doubtful Obstructive Defects	3	6	0	0	0	0	5	3
TOTALS	50	100	98	100	45	100	176	100

TABLE 4.XXVIII

Radiographic Abnormality in Lung Function Categories

Radiographic Profusion of small irregular opacities	Lung Function Assessment									
	Normal		Transfer Defect		Restricted TLC		Transfer Defect + Restricted TLC		Obstructive Defect	
	No.	%	No.	%	No.	%	No.	%	No.	%
0/0	189	72	11	44	16	47	6	38	8	73
0/1	62	23.6	11	44	13	38	2	12	3	27
1	11	4	2	8	4	12	6	38	-	-
2	1	0.4	1	4	1	3	2	12	-	-
TOTAL	263	100	25	100	34	100	16	100	11	100

NOTE. "Other defects", and "doubtful defects" shown in the previous Table are omitted from this Table.

TABLE 4.XXIX

Physical Signs by Occupational Group and Years of Exposure

Sprayers					Laggers			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	4	22	21	3	55	16	22	5
No. with rales	1	16	10	2	1	3	11	3
No. with rhonchi	0	4	4	0	7	4	4	1
No. with clubbing	0	6	8	1	3	2	2	2
No. with Grade 3 + dyspnoea	1	7	4	1	4	2	5	3
Sailmakers, Storemen & Masons					Intermittently Exposed			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	31	7	5	2	53	61	37	25
No. with rales	3	3	1	1	0	5	6	6
No. with rhonchi	2	0	0	1	3	5	6	2
No. with clubbing	1	0	0	0	1	4	5	2
No. with Grade 3 + dyspnoea	1	0	0	0	0	4	3	4

TABLE 4.XXX

X-ray Categories by Occupational Groups and Years of Exposure

Sprayers					Laggers			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	4	22	21	3	55	16	22	5
No. 0/0	4	10	8	0	44	10	8	1
No. 0/1	0	8	8	0	9	6	10	2
No. 1	0	2	4	3	2	0	4	1
No. 2	0	2	1	0	0	0	0	1
Sailmakers, Storemen & Masons					Intermittently Exposed			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	31	7	5	2	53	61	37	25
No. 0/0	24	3	3	1	47	41	21	15
No. 0/1	3	3	0	0	6	18	14	9
No. 1	3	1	1	1	0	2	2	1
No. 2	1	0	1	0	0	0	0	0

X-ray Categories Profusion of Small Opacities

TABLE 4.XXXI

Lung Function Categories in 10 Year Periods of Exposure

	Sprayers				Laggers			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	4	22	21	3	55	16	22	5
Normal	1	8	13	1	41	9	10	1
Transfer Defect	1	3	4	1	7	1	3	0
TLC Reduced	0	5	2	0	5	4	6	2
Transfer Defect + TLC Reduced	1	3	1	1	2	2	3	2
Obstructive Defect	1	1	0	0	0	0	0	0
Other Doubtful Defects	0	2	1	0	0	0	0	0
	Sailmakers, Storemen & Masons				Intermittently Exposed			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
No. of Men	31	7	5	2	53	61	37	25
Normal	26	5	2	1	49	51	26	19
Transfer Defect	0	0	1	0	3	1	0	0
TLC Reduced	1	1	1	0	0	4	1	2
Transfer Defect + TLC Reduced	1	1	0	0	0	0	0	0
Obstructive Defect	1	0	0	0	1	1	4	2
Other Doubtful Defects	2	0	1	0	0	4	6	2

TABLE 4.XXXII

Mean Values for Lung Function Tests in 10 Year Periods of Exposure

Years of Exposure	0-9	10-19	20-29	30+
Number of Men	143	106	85	35
FVC	4.34	3.87	3.61	3.07
$FEV_{1.0}$	3.41	2.94	2.70	2.21
RV	1.66 **	1.76	1.90	2.03 *
TLC	6.30 **	5.89	5.75	5.45 *
T_L	30.1 **	26.5	25.5	24.6 *
T_L1	29.0 **	25.2	24.0	21.6
D_M	47.1 **	40.6 *	40.1	38.6 **
Hyponychial Angle°	184	184	186	186
Age (Years)	36.4	45.3	51.7	58.1
Height (cms)	172.1	170.5	169.7	167.3

Lung Volumes expressed in litres BTPS.

T_L, T_L1, D_M in mlCO/min/mmHg.

* One man in this group did not perform this test

** Two men in this group did not perform this test

TABLE 4.XXXIII

Mean Values for Lung Function Tests of Laggers and Intermittently Exposed in 10 Year Periods of Exposure

	Intermittently Exposed				Laggers			
Years of Exposure	0-9	10-19	20-29	30+	0-9	10-19	20-29	30+
Number of Men	53	61	37	25	55	16	22	5
FVC	4.60	4.19	3.65	3.08	4.22	3.84	3.47	3.09
$FEV_{1.0}$	3.68	3.22	2.71	2.24	3.34	2.93	2.63	2.36
RV	1.65*	1.78	2.03	2.11*	1.52*	1.44	1.68	1.64
TLC	6.63*	6.29	5.99	5.60*	5.90*	5.40	5.30	5.07
T_L	33.1*	28.9	27.1	26.5*	27.7*	25.2	23.7	22.4
T_L1	31.7*	27.3	25.1	23.0	27.3*	25.2	22.8	21.0
D_M	52.7*	44.1	42.2	41.2**	43.6*	37.6	38.1	34.5
Hyponychial Angle°	183	183	185	184	186	183	187	191
Age (Years)	32.5	42.1	51.4	57.9	37.0	45.1	51.1	57.0
Height (cms)	172.3	170.9	169.8	166.2	171.8	169.9	169.6	172.6

Lung Volumes expressed in litres BTPS

T_L, T_L1, D_M in mlCO/min/mmHg.

*** One man in this group did not perform this test**

**** Two men in this group did not perform this test**

TABLE 4.XXXIV

Symptom and Sign Complexes in Radiographic Categories

Profusion of Opacities	0/0				0/1		1+2	
Pleural Abnormality	-		+		+-		+-	
Sign and Symptom Group	No.	%	No.	%	No.	%	No.	%
1	24	13	18	29	38	39	20	64
2	12	7	5	9	13	13	2	6
3	144	80	38	62	46	48	9	30
TOTALS	180	100	61	100	97	100	31	100

Group 1. Men with rales; rales + clubbing; rales + dyspnoea; rales, clubbing + dyspnoea; dyspnoea; clubbing; rales + rhonchi + clubbing.

Group 2. Men with rhonchi; rhonchi + clubbing; rhonchi + dyspnoea; rhonchi + clubbing + dyspnoea.

Group 3. Men without abnormal symptoms or signs.

TABLE 4.XXXV

Mean Values for Standardized Ventilation in X-ray Categories

Profusion of Opacities	0/0		0/1		1		2	
Pleural Abnormality	-	+	-	+	-	+	-	+
Number of Men	146	46	44	28	7	8	1	2
Standardized Ventilation litres per minute	33.4	35.6	35.9	38.2	40.1	39.7	48.3	47.5

TABLE 4.XXXVI

Mean Values for Exercise Ventilation (EV), Standardized Ventilation (SV) and for Transfer Factor (T_L) in Grades of Dyspnoea

Grade of Dyspnoea	1	2	3+
Number of Men	176	80	13
Exercise Ventilation (litres per minute)	32.5	35.7	41.0
Standardized Ventilation (litres per minute)	33.0	36.8	44.2
Transfer Factor ml/min/mmHg	30.3	26.2	22.0

TABLE XXXVII

Eleven Men with Calcified Pleural Plaques

Occupations	Number of Men
Sprayers	3
Laggers	2
Intermittently Exposed	6

Symptoms and Signs	Number of Men
Rales	4
Clubbing	2
Dyspnoea	2
None	3

Mean Values	
Age (Years)	52
Height (cms)	170.5
Weight (Kg)	70.6
Years of Exposure	23.27
Years since 1st Exposure	25.72
Hyponychial Angle°	183.1

Mean Values	
FVC	3.66 l
$FEV_{1.0}$	2.72 l
RV	1.73 l
TLC	5.47 l
T_L	24.53 mlCO
$T_L 1$	23.64 mlCO
D_M	37.10 mlCO

Lung Function Category	Number of Men
Normal	6
Transfer Defect	1
Restrictive Defect	3
Doubtful Obstructive Defect	1

X-ray Category		
Profusion of Opacities	Pleural Abnormality	
	Absent	Present
	Number of Men	
0/0	0	4
0/1	0	4
1	0	3
2	0	0

TABLE XXXVIII

36 Men Accepted by Pneumoconiosis Medical Panel as having Asbestosis

Occupations	Number of Men	Symptoms and Signs	Number of Men
Sprayers	15	Rales	32
Laggers	17	Rhonchi	3
Sailmaker	1	Clubbing	11
Intermittently Exposed	3	Dyspnoea	17

Mean Values		Mean Values	
Age (Years	55	FVC	3.09 l
Height (cms)	170.4	$FEV_{1.0}$	2.09 l
Weight (Kg)	71.1	RV	1.72 l
Years of Exposure	22.3	TLC	5.01 l
Years since 1st Exposure	23.9	T_L	19.10 mlCO
Hyponychial Angle°	190	T_L 1	18.09 mlCO
		D_M	30.20 mlCO

Lung Function Category	Number of Men
Normal	6
Transfer Defect	6
Restrictive Defect	10
Restrictive + Transfer Defect	11
Doubtful Restrictive or Transfer Defects	3

X-ray Category		
Profusion of Opacities	Pleural Abnormality	
	Absent	Present
	Number of Men	
0/0	1	7
0/1	5	9
1	2	8
2	0	4

APPENDIX (Table I)

Data for men excluded from analysis because of exposure to other dust (See Table 4.VI)

Laggers (Numbers in brackets refer to numbers in Table)							
Case No.	(1)	(2)	(3)	(4)	(5)	(6)	(7)
X-ray Category Opacities	0/0	0/1	0/0	0/1	1	0/1	0/1
Pleural Abnormality	-	+	+	-	+	-	-
FVC	5.23	2.83	3.00	4.19	2.93	4.90	5.59
$FEV_{1.0}$	4.53	2.25	1.69	3.65	2.42	3.85	4.08
FEV/FVC%	86.60	79.00	56.30	87.10	82.50	78.50	72.90
SVC	5.40	3.25	3.29	4.31	2.92	5.00	5.97
RV	2.48	2.07	1.69	1.18	1.16	1.41	2.16
TLC	7.98	5.32	4.98	5.49	4.08	6.41	8.13
RV/TLC%	32.30	39.00	33.90	21.50	28.40	21.90	26.60
$T_L CO$	31.20	24.30	19.00	25.90	14.90	26.50	31.10
$T_L 1$	31.20	20.30	17.90	27.60	13.80	27.40	31.30
Dm	45.50	38.30	27.10	40.60	18.10	42.00	43.40
Hyponychial Angle	197	180	192	184	203	191	189
Years of Exposure	3	13	28	17	16	16	9
Age (Years)	46	54	60	45	44	40	43
Height (cms)	176	170	178	183	173	174	173
Weight (Kg)	73	60	74	92	73	93	67
Lung function x Category	N	N	R + T	N	RT	N	N
Cough & phlegm 3 months	-	+	+	-	+	+	+
Chest Illness	-	-	+	-	+	+	+
Smoking Category	15-24g	5-14g	5-14g	Ex	15-24g	15-24g	15-24g
Dyspnoea Grade	2	1	0	2	3	1	2
Rales	-	-	+	-	+	-	-
Rhonchi	-	-	-	-	-	-	-
Clubbing	-	-	-	-	+	+	-
Asbestos corns	+	+	+	+	+	-	+
Asbestos in sputum	+	+	+	-	+	+	+

APPENDIX (Continued)

	Intermittent Exposure	Sailmakers, Storemen & Masons		
Case No.	(8)	(9)	(10)	(11)
X-ray Category Opacities	0/0	0/0	0/1	0/0
Pleural Abnormality	-	+	+	-
FVC	3.54	2.13	3.98	3.15
$FEV_{1.0}$	2.96	1.63	3.03	2.68
FEV/FVC%	83.60	76.50	76.10	85.10
SVC	4.17	2.75	4.12	3.55
RV	2.16	2.81	1.20	1.26
TLC	6.77	5.56	5.32	4.81
RV/TLC%	38.50	50.60	22.60	26.20
$T_L CO$	30.40	24.30	23.70	16.00
$T_L 1$	26.30	19.40	23.50	19.70
Dm	45.50	55.10	38.70	-
Hyponychial Angle	177	189	Not Measured	180
Years of Exposure	10	44	18	3
Age (Years)	54	66	59	45
Height (cms)	172	168	172	172
Weight (Kg)	66	65	73	59
Lung function x Category	N	N	R	RT
Cough & phlegm 3 months	-	-	-	-
Chest Illness	-	-	-	+
Smoking Category	NON	25+	5-14g	5-14g
Dyspnoea Grade	1	1	2	1
Rales	-	-	-	-
Rhonchi	-	-	-	-
Clubbing	-	-	-	-
Asbestos corns	-	-	-	-
Asbestos in sputum	+	-	-	+

APPENDIX

Men excluded from Analysis because of Exposure to other Dust

Table 4.VI (page 207) shows that the asbestos exposure for most of these men was much longer than their exposure to other dust. The clinical and lung function data are recorded in Table I of this Appendix. Three of the laggers (Case Nos. 2, 3 and 5) have been accepted as having asbestosis by the Pneumoconiosis Medical Panel. Case No. 2 had an extensive pleural reaction, Case No. 3 hyaline plaques, and Case No. 5 had extensive nodulation and pleural abnormalities. The last case was quite severely disabled by dyspnoea and had very marked finger clubbing.

The remaining men were relatively normal.

APPENDIX

Age and Cause of Death of Men Trained as Asbestos Sprayers

Cause of Death	Age	Year of Death
1. Carcinoma of lung Carcinomatosis	42	1954
2. Coronary thrombosis	45	1948
3. Carcinoma of larynx	58	1955
4. Carcinoma of stomach	58	1961
5. Pulmonary tuberculosis	61	1953
6. Carcinoma of stomach	64	1956
7. Motor accident	58	1960
8. Myocardial infarction	42	1965
9. Auricular fibrillation Hyperpiesis	68	1964
10. Carcinomatosis Carcinoma of prostate	64	1965
11. Carcinomatosis Carcinoma of colon	65	1962
12. Cerebral embolism Mitral regurgitation Acute rheumatism	46	1948

MEDICAL RESEARCH COUNCIL QUESTIONNAIRE ON RESPIRATORY SYMPTOMS (1960)

DEVONPORT DOCKYARD ASBESTOS SURVEY

QUESTIONNAIRE ON RESPIRATORY SYMPTOMS (1960)

(Approved by Medical Research Council Committee on Aetiology Bronchitis)

SURVEY or HOSPITAL .....

NAME .....
(Surname)

.....
(First Names)

ADDRESS .....

.....

Other Registration data:-

Serial No. .....

Day Month Year
figures last 2 digits

Date of Interview ....

Date of Birth ....

(Sex) - M F

Age ....

S M W

Civil State ....

Race .....

Standing height .....

Sitting height .....

Weight .....

Interviewer .....

Use actual wording of each question. Put X in appropriate square after each question. When in doubt, record 'No'.

COUGH	Yes	No	
Do you usually cough first thing in the morning/on getting up* in the winter? Count a cough with first smoke or on 'first going out of doors'. Exclude clearing throat or a single cough.	....	....	1
Do you do this in the summer?	....	....	2
Do you usually cough during the day or at night in the winter? Ignore an occasional cough.	....	....	3
Do you do this in the summer?	....	....	4

If 'Yes' to any questions 1-4:-

	Yes	No	
Do you cough like this on most days for a much as three months each year?	....	....	5

PHLEGM or alternative word to suit local custom

	Yes	No	
Do you usually bring up any phlegm from your chest first thing in the morning/on getting up* in the winter? Count phlegm with the first smoke or on 'first going out of doors'. Exclude phlegm from the nose. Count swallowed phlegm.	....	....	6
Do you do this in the summer?	....	....	7
Do you usually bring up any phlegm from your chest during the day, or at night, in the winter? Accept twice or more.	....	....	8
Do you do this in the summer?	....	....	9

If 'Yes' to any questions 6-9:-

	Yes	No	
Do you bring up phlegm like this on most days for as much as three months each year?	....	....	10

If 'Yes' to questions 5 or 10:-

**How long have you had this phlegm/cough? Ask about cough if no phlegm. — 2 years or less	....	....	11a
**How long have you had this phlegm/cough? Ask about cough if no phlegm. — More than 2 years	....	....	11b

*These words are for subjects who work by night.
**For subjects who usually have phlegm.

In the past three years, have you had a period of (increased**) cough and phlegm lasting for 3 weeks or more? — No			.... 12a
In the past three years, have you had a period of (increased**) cough and phlegm lasting for 3 weeks or more? — Yes — Only one period			.... 12b
In the past three years, have you had a period of (increased**) cough and phlegm lasting for 3 weeks or more? — Yes — Two or more periods			.... 12c
**For subjects who usually have phlegm			
**Have you ever coughed up blood? — No			.... 13a
**Have you ever coughed up blood? — Yes — Streaks			.... 13b
**Have you ever coughed up blood? — Yes — More			.... 13c

Greatest amount at one time

If 'Yes to 13:- When was this? 13d
each year of occurrence

BREATHLESSNESS

Questions refer to average condition in winter

If disabled from walking by any condition other than heart or lung disease put X here — and leave questions 14a-c unasked	....		14

	Yes	No	
Are you ever troubled by shortness of breath, when hurrying on the level or walking up a slight hill?	....	....	14a

If 'No', grade 1. If 'Yes' proceed to the next question.

	Yes	No	
Do you get short of breath walking with other people at an ordinary pace on the level?	....	....	14b
If 'No', grade is 2. If 'Yes', proceed to next question.			
Do you have to stop for breath when walking at your own pace on the level?	....	....	14c
If 'No', grade is 3. If 'Yes' proceed to next question.			
Are you short of breath on washing or dressing?	....	....	14d
If 'No', grade is 4. If 'Yes', grade is 5.			

WHEEZING

Does your chest ever sound wheezy or whistling?	....	....	15
If 'Yes' to 15:-			
Do you get this with colds?	....	....	15a
Do you get this occasionally apart from colds?	....	....	15b
Do you get most days or nights?	....	....	15c
**Have you ever had attacks of shortness of breath with wheezing?	....	....	16
If 'Yes' to 16:- When did you first get these attacks? Before age 30	....	....	16a
If 'Yes' to 16:- When did you first get these attacks? At or after age 30	....	....	16b
Do you still get these attacks?	....	....	16c

EFFECT OF WEATHER

Does the weather affect your chest?	....	....	17
Only record 'Yes' if adverse weather definitely and regularly causes chest symptoms.			
If 'Yes to 17:-			
Does foggy weather affect it?	....	....	17a
Does damp weather affect it?	....	....	17b
Does cold weather affect it?	....	....	17c
Does hot weather affect it?	....	....	17d
Does any other sort of weather affect it?	....	....	17e
If 'Yes' to 17e: Specify			
If 'Yes' to 17a-e: Does this weather make you short of breath?	....	....	17f

NASAL CATARRH

	Yes	No	
Do you usually have stuffy nose or catarrh at the back of your nose in the winter?	....	....	18
Do you have this in the summer?	....	....	19

If 'Yes' to 18 or 19:-

Do you have this on most days for as much as 3 months each year?	....	....	20

CHEST ILLNESSES

During the past 3 years have you had any chest illness which has kept you off work, indoors, at home or in bed?	....	....	21

If 'Yes' ask details of each illness:-

Year	Duration of incapacity		Increased Phlegm		Doctor's Diagnosis
	Less than 1 week	1 week or more	Yes	No	

**PAST ILLNESSES

		Age of occurrance	Yes	No	
Have you ever had:-	Bronchitis?	.....	....	....	22
Put each age at which illnesses occurred on dotted lines.	Pneumonia?	.....	....	....	23
	Pleurisy?	.....	....	....	24
	Pulmonary Tb.?	.....	....	....	25
If more than 3 attacks put 'recurrent' after age at first attack.	Bronchial Asthma?	.....	....	....	26
	If 'Yes' to 26: Age started			....	26a
	Age stopped			....	26b

	Age of occurrance	Yes	No	
Other chest illness?		....	....	27

If 'Yes' to 28, specify:

.....

.....

Were you gassed in World War I? 29

Have you ever been badly affected by gas or fumes at work? 30

If 'Yes' to 29 or 30, give details: .....

.....

---

*DIAGNOSIS (For doctors only)

In your opinion does the subject have:-

Chronic Bronchitis?	....	....	31
Bronchial Asthma?	....	....	32
Emphysema?	....	....	33
Other Chest Disease?	....	....	34

If 'Yes' to 34, specify: .....

TOBACCO SMOKING	Yes	No	
Do you smoke? Record 'Yes' if regular smoker up to one month ago.	....	....	35

If 'No' to 35:-

Have you ever smoked? Record 'No' if subject has never smoked as much as one cigarette a day, or 1 oz. of tobacco a month, for as long as one year.	....	....	36

If 'Yes' to 35 or 36, - Fill in figures below:

	Amount smoked		
	Now	Previously	
Cigarettes/day (average, including weekends)			37a
Ozs. tobacco/week (handrolled)			37b
Ozs. tobacco/week (pipe)			37c
Cigars/week (large)			37d
Cigars/week (small)			37e

Age started regular smoking 38a

Age stopped regular smoking 38b

If smoking stopped or reduced, give reasons 38c

.....

OCCUPATIONAL AND RESIDENTIAL HISTORY

Work systematically from birth forwards for residence and from leaving school for occupation, ensuring that no periods are omitted. Record actual year's job and residence started and stopped. Under 'Residence' record actual towns lived in (put outskirts' if this applies). For villages and rural areas record county; foreign residence only the country; seamen as 'at sea'.

Give full details of any periods of work in coal or other mines, foundries potteries, cotton/flax/hemp, asbestos, other dusty jobs and of any exposure to irritating gas and chemical fumes.

Dates	Ages	Industry	Job (actual occupation)	Residence
(1890-)				
(1900-)				
(1910-)				
(1920-)				
(1930-)				
(1940-)				
(1950-)				
(1960-)				

SPUTUM

Nil	Negative	Fibres	Asbestos Bodies	Bodies & Fibres
0	1	2	3	4

LUNG FUNCTION

Number	FVC	$FEV_{1.0}$
1		
2		
3		
4		
5		
Mean of last three		

FEV/FVC% =

RV .....

TLC .....

RV/TLC .....

TL .....

TL .....

CLINICAL

1. General
2. Hb.
3. VCS
4. BP
5. Cyanosis
6. RS
7. Rales
8. Rhonchi
9. Expansion
10. Clubbing
11. Asbestos Corns
12. Comments

Year of 1st exposure	
Year of last exposure	
Period of exposure (years)	
Type of exposure	
Continuous	Heavy
	Light
Intermittent	Heavy
	Light
Comments	

X-RAY

U.I.C.C./CINCINNATI CLASSIFICATION OF RADIOGRAPHIC APPEARANCE OF PNEUMOCONIOSES

Survey		ROUNDED SMALL OPACITIES			IRREGULAR SMALL OPACITIES			LARGE OPACITIES		PLEURAL THICKENING					ILL DEFINED DIAPHRAGM	ILL DEFINED CARDIAC OUTLINE	PLEURAL CALCIFICATION					Reader _____ Date _____	
Number	Quality	Type	Profusion	Zones R L	Type	Profusion	Zones R L	Well defined	Ill defined	Costophrenic angle	Diffuse	Plaques	Grade	None			Diaphragm	Wall	Other	Grade	None	Symbols	Comments
			/			/																	
			/			/																	
			/			/																	
			/			/																	
			/			/																	
	+	p	0/- 0/0 0/1	Check zones involved	s	0/- 0/0 0/1	Check zones involved	0		0			0		0	0				0		0	
	±	q	1/0 1/1 1/2		t	1/0 1/1 1/2		A		R	R		1		R	1	R			1		ca es	
	±	r	2/1 2/2 2/3		u	2/1 2/2 2/3		B		L	L		2	0 if none	L	2	L			2	0 if none	co od	
	U/R		3/2 3/3 3/4			3/2 3/3 3/4		C					3			3				3		cp tba	

Details of calculations for the determination of lung volumes, transfer factor and its sub-divisions.

Examples are shown at the end of the calculation of the data sheet prepared to transfer the data to the computer, and computer print outs for the entire calculation, and for a smaller programme which does not include the calculation of Dm and Vc.

ORDER OF DATA FOR COMPUTER

1	Subject No.	22	25	BHT cms	7.50
2	Weight Kg	72	26	0	0
3	Standing Ht. cms	176	27	Vol. insp. cms	14.81
4	Sitting Ht. cms	90	28	Exp. He	10.71
5	Day	3	29	Exp. O_2	85.3
6	Month	7	30	M He_1	7.19
7	Year	67	31	M He_2	7.08
8	Temp. ^oC	20	32	MCO_1	49.7
9	BP mm Hg	765	33	MCO_2	49.1
10	Initial Helium (H_1)	11.40	34	Exp. CO	49.7
11	Final Helium (H_2)	6.45	35	Age (years)	35
12	IC cms	10.29	36	Vs cms	2.48
13	ERV cms	6.71	37	Vw cms	2.13
14	Final vol. cms	12.84	38	Insp. Helium	14.39
15	Initial vol. cms	12.20	39	BHT cms	7.60
16	O_2 uptake	1.80	40	0	0
17	Hb %	100	41	Vol. insp. cms	13.64
18	Bt CO	8.3	42	Exp. Helium	10.08
19	Bt O_2	98.4	43	Exp. O_2	17.2
20	Bt MCO	18.4	44	M He_1	6.22
21	Bt MHe	0.90	45	M He_2	5.88
22	Vs cms	1.88	46	MCO_1	46.1
23	Vw cms	2.36	47	MCO_2	44.0
24	Insp. Helium	12.45	48	Exp. CO	45.4

	Calculation of TLC and Sub-divisions	Data
F	Functional Residual Capacity (litres ATPS)	
H_1	Initial He % reading (i.e. at "switch in")	11.40
H_2	Final He % reading (at equilibrium)	6.45
V_1	Vol. of gas in spirometer before "switch in".	12.20 cms
V_2	Vol. of gas in spirometer at end exp. level	12.84 cms
D_1	Deadspace of Machine } CONSTANTS	1.76 l
D_2	Deadspace of Mouthpiece }	0.291 l
BP	Barometric Pressure mm Mercury	765
Temp.	Temperature ^{o}C	20
PW	Water vapour pressure	

i.e. $e^{(0.051647 \times t + 2.015)} - 3.5$

Correction of Data

H_1 and H_2 (He Meter scale non-linear correction for DDS meter only)

x = Observed He % reading

y = Corrected He % reading

* Correction obtained by calibration against Helium Kathorometer at PRU.

$y = 1.072 (x - 0.2)$

$H_1 = (11.40 - 0.2)\ 1.072 = 12.0064$

$H_2 = (6.45 - 0.2)\ 1.072 = 6.7000$

Convert V_1, V_2 cms ⟶ litres ATPS

$V_1 = 12.2 \div 3$ (Spirometer displacement = 3 cms/litre; i.e. $\frac{V \text{ cms}}{3.00 \text{ cm/l}} = V.\ l$) = 4.0667 litres

$V_2 = 12.84 \div 3$ = 4.2800 litres

$$F = \frac{H_1}{H_2}(V_1 + D_1 - D_2) - (V_2 + D_1)$$

$$= \frac{12.0064}{6.7000}(4.0667 + 1.76 - 0.291) - (4.2800 + 1.76)$$

$$= 9.9200 - 6.0400$$

$$= 3.8800 \text{ ATPS}$$

Convert 'F' to BTPS

$$3.8800 \times \frac{310}{(273 + 20)} \left\{ \frac{765 - e(0.051647 \times 20 + 2.015) - 3.5}{765 - 47} \right\}$$

FRC = 3.8800 x 1.1014 = 4.2734 litres BTPS

FRC	Functional Residual Capacity
ERV	Expiratory Reserve Volume
RV	Residual Vol. (FRC-ERV)
IC	Inspiratory Capacity
VC	Vital Capacity (ERV + IC)
TLC	Total Lung Capacity (RV + IC + ERV)
RV/TLC %	Ratio %

ERV	= 6.71 ÷ 3 x 1.1014	= 2.4635 l BTPS
RV	= 4.2734 - 2.4635	= 1.8099 l "
IC	= 10.29 ÷ 3 x 1.1014	= 3.7778 l "
VC	= 2.4635 + 3.7778	= 6.2413 l "
TLC	= 1.8099 + 3.7778 + 2.4635	= 8.0512 l BTPS
RV/TLC %	= 22.4799 %	

	Transfer Factor Single Breath Carbon Monoxide (low tension oxygen)	Data
Va^1	Breath-holding alveolar Vol. (1 BTPS)	
V insp.	Vol. insp. (1 BTPS) 13.64 ÷ 3 x 1.1273 (Conversion Factor = $\frac{310}{273 + t} \times \frac{B}{B - 47}$)	5.1255
Vs	Vol. of sample (no need for corr. to BTPS) 2.48 ÷ 3	0.8267
BHT	Breath-holding time 7.60 ÷ 0.865 cm/sec	8.7861
HI	He % (dry) inspired	14.39
Hs	He % (dry) in sample	10.08
MCO_1	Matching CO (larger value)	46.1
MCO_2	Matching CO	44.0
CO exp	Expired CO	45.4
$M\ He_1$	Matching He (larger value)	6.22
$M\ He_2$	Matching He	5.88
Ds	Anatomical Deadspace	0.15
D	Deadspace sample bag	0.059
a	Fractional conc. CO_2 in exp. gas (removed) 6% for 10 sec BHT $\left(\frac{6}{100}\right)$	0.06

Correct all He readings *(For DDS meter only).

$H_1 = (14.39 - 0.2)\ 1.072 = 15.2117$

$Hs = (10.08 - 0.2)\ 1.072 = 10.5914$

$MH_1 = (6.22 - 0.2)\ 1.072 = 6.4534$

$MH_2 = (5.88 - 0.2)\ 1.072 = 6.0890$

$$Va1 = V\ insp - DS \left\{ \frac{HI}{\frac{Vs + D}{Vs}\ (1 - a)\ Hs} \right\}$$

$$= 5.1255 - 0.15 \left(\frac{15.2117}{\left(\frac{0.8267 + 0.059}{0.8267} \right) (1.00 - 0.06 \times 10.5914)} \right)$$

$\underline{Va^1 = 7.0956 \text{ l BTPS}}$

$RV^1 = Va^1 - V \text{ insp} = \underline{1.9701} \text{ l BTPS}$

TL^1 Diffusing capacity of lung (ml/min/mmHg)

$$TL^1 = \frac{160 \times Va^1}{BHT} \log_{10} \frac{Hs}{MHe}$$

$$MHe = H_2 + \left(\frac{(MHe_1 - MHe_2)}{(CO_1 - CO_2)} \right) CO \text{ exp.} - CO_2$$

$$= 6.0890 + \left(\frac{6.4534 - 6.0890}{46.1 - 44.0} \right) 45.4 - 44.0$$

$MHe = 6.3319$

$$TL^1 = \frac{160 \times 7.0956}{8.7861} \log_{10} \frac{10.5914}{6.3319}$$

$TL^1 = 129.2150 \times 0.2235$

$\underline{TL^1 = 28.8796}$ ml/min/mmHg STPD

Va Full alveolar volume (l BTPS)

Va = V insp. + RV

$= 5.1255 + 1.8099 = \underline{6.9354}$

$$TL = \frac{160 \times 6.9354}{8.7861} \times 0.2235$$

$\underline{TL = 28.2275}$ ml/min/mmHg STPD

	Transfer Factor Single Breath Carbon Monoxide (High Tension Oxygen)	Data
Va^1	Breath-holding alveolar Vol. (1 BTPS)	
V insp.	Vol. insp. (1 BTPS) 14.81 ÷ 3 x 1.1273 (Conversion factor = $\frac{310}{273 + t} \times \frac{B}{B-47}$)	5.5651
Vs	Vol. of sample (no need for corr. to BTPS) 1.88 ÷ 3	0.6267
BHT	Breath-holding time 7.50 ÷ 0.865 cm/sec	8.6705
HI	He % (dry) inspired	12.45
*Hs	He % (dry) in sample (to be corr. for O_2/N_2)	10.71
MCO_1	Matching CO (larger value)	49.7
MCO_2	Matching CO	49.1
CO exp.	Expired CO	49.7
MHe_1	Matching He (larger value)	7.19
MHeS	Matching He	7.08
Ds	Anatomical Deadspace	0.15
D	Deadspace Sample Bag	0.059
a	Fractional conc. CO_2 in exp. gas (Removed) 6% for 10 sec. BHT $\left(\frac{6}{100}\right)$	0.06

Correct all He readings * (For DDS Meter only)

HI = (12.45 - 0.2) 1.072 = 13.1320

Hs = (10.71 - 0.2) 1.072 = 11.2667

MH_1 = (7.19 - 0.2) 1.072 = 7.4933

MH_2 = (7.08 - 0.2) 1.072 = 7.3754

Correct He % in sample for effect of O_2/N_2

$$He\ \% = \frac{(O_2 - 21)^x}{y}$$

x, y } from calibration of DDS He meter.

$$= \frac{(85.3 - 21)^{0.8608}}{25.29}$$

$$= 1.4243 \qquad \therefore \quad \underline{HeS = 9.8424}$$

	Calculation of back tension of carbon monoxide	Data
Bt CO exp.	CO sample from rebreathing	8.3
Bt MHe	Matching He = (0.9-0.2) 1.072	0.7504
Bt MCO	Matching CO	18.4
Bt	Back Tension	
$R_1\ O_2$	O_2 rebreathed	98.4
Cs, O_2	Exp. O_2 from sample concentration	85.3

$$Bt\ He = \frac{Bt\ CO}{Bt\ MCO} \times Bt\ MHe = \frac{8.3}{18.4} \times 0.7504 = \underline{0.3385}$$

$$Bt = 0.3385 \times \frac{85.3}{98.4}$$

$$\underline{Bt = 0.2934}\ \text{mls/min/mmHg}$$

$$Va^1 = V\ insp. - Ds \left\{ \frac{HI}{\frac{Vs + D}{Vs}\ (1 - a)\ Hs} \right\}$$

$$= 5.5651 - 0.15 \left\{ \frac{13.1320}{\frac{0.6267 + 0.059}{0.6267}\ (0.94 \times 9.8424)} \right\}$$

$$\underline{Va^1 = 7.0250}$$

$$MHe = 7.3754 + \left\{ \frac{7.4933 - 7.3754}{49.7 - 49.1} \right\} 49.7 - 49.1$$

$$\underline{MHe = 7.4933}$$

$$TL^1 = \frac{160 \times Va^1}{BHT} \times \log_{10} \left\{ \frac{Hs - Bt}{MHe - Bt} \right\}$$

$$TL^1 = \frac{160 \times 7.0250}{8.6705} \times \log_{10} \frac{(9.8424 - 0.2934)}{(7.4933 - 0.2934)}$$

$TL^1 = 15.8855$ ml/min/mmHg STPD

Va = V insp. + RV

= 5.5651 + 1.8099 = 7.3750

$$TL = \frac{160 \times 7.3750}{8.6705} \log_{10} \frac{(9.8424 - 0.2934)}{(7.4933 - 0.2934)}$$

TL = 16.6769 ml/min/mmHg STPD

Calculation for predicted TL^1 (Cotes)

TL^1 = 33 x Height (Meters) - 0.24 (age) - 17.2 = 32.48

	Calculation of Alveolar Oxygen Tension	Data
PA, O_2	Alveolar Tension High Tension Oxygen	
PA, Air	Alveolar Tension Low Tension Oxygen	
Vs, O_2	Vol. of sample High O_2	0.6267
Vs, Air	Vol. of Sample Low O_2	0.8267
CS, O_2	Fractional conc. O_2 = $\frac{85.3}{100}$ (Exp. O_2)	0.853
CS, Air	Fractional conc. Air = $\frac{17.2}{100}$ (Exp. O_2)	0.172
B	Barometric Pressure mmHg	765
a	Fractional conc. CO_2 (Removed) 1-0.06	0.94
D	Deadspace sample bag	0.059
PW	Vapour Pressure BTPS mmHg	47
Air	Fract. conc. of O_2 in Air	0.2093

$$PA, O_2 = (B - 47)\left(\frac{(Vs + D)(C.94.CS) - D(0.2093)}{Vs}\right)$$

$$= (765 - 47)\left(\frac{(0.6267 + 0.059)(0.94 \times 0.853) - 0.059(0.2093)}{0.6267}\right)$$

PA, O_2 i.e. P_2 = 615.8286 mmHg

$$PA, Air = (765 - 47)\left(\frac{(0.8267 + 0.059)(0.94 \times 0.172) - 0.0123)}{0.8267}\right)$$

PA, Air i.e. P_1 = 113.6594 mmHg

	Calculation of Dm & Vc	Data
Dm	Diffusing capacity of alveolar membrane	
Vc	Vol. of blood in alveolar capillaries	
P_1	Air Tension in alveoli	113.6594
P_2	O_2 Tension in alveoli	615.8286
D_1	Transfer Factor Air	28.2275
D_2	Transfer Factor O_2	16.6769

		Data
V, O_2	Oxygen uptake ml/min	1.80
Hb	Haemoglobin %	100
θ	Rate of combination of O_2 with Hb	0.57
dCO	Diffusivity of CO relative to O_2	1.23
C	CONSTANT	57.89

$$V, O_2 = 1.80 \text{ cm/2 min}$$

$$= 0.90 \text{ cm/min}$$

3.0 cms $\longrightarrow$ 1 litre

$$= \frac{0.90 \times 1000}{3} = 300 \text{ ml/min ATPD}$$

$$\text{Convert to STPD} = \frac{273}{273 + t} \times \frac{BP}{760} = 0.9378$$

$$V, O_2 = 300 \times 0.9378 = 281.34 \text{ ml/min}$$

$$Dm = \frac{D_1 D_2 (P_2 - P_1) - \frac{V, O_2}{1.23} (D_1 - D_2)}{D_2 P_2 - D_1 P_1 - 57.89 (D_1 - D_2)}$$

$$= \frac{28.2275 \times 16.6769 (615.8286 - 113.6594) - \frac{281.34}{1.23} (28.2275 - 16.6796)}{(16.6769 \times 615.8286) - (28.2275 \times 113.6594) - 57.89 (28.2275 - 16.6769}$$

$$Dm = \frac{233752.7529}{6393.1271} = \underline{36.5631 \text{ ml/min/mmHg STPD}}$$

$$Vc = \frac{0.57 (P_2 - P_1) D_1 D_2}{Hb\ \% (D_1 - D_2)}$$

$$= \frac{0.57 (615.8286 - 113.6594) 28.2275 \times 16.6769}{100 (28.2275 - 16.6769)}$$

$$Vc = \frac{134745.0025}{1155.06} = \underline{116.6563 \text{ mls.}}$$

$$\frac{Dm}{Vc} = \underline{0.3134}$$

Data

Dm^1 Diffusing capacity of alveolar membrane using TL^1

Vc^1 Vol. of blood in alveolar capillaries using TL^1

D_1^1 Transfer Factor (AIR) 28.8796

D_2^1 Transfer Factor (O_2) 15.8855

$$Dm^1 = \frac{28.8796 \times 15.8855 \times 502.1692 - 2972.1626}{(15.8855 \times 615.8286) - (28.8796 \times 113.6594) - 57.89 \times 12.9941}$$

$$Dm^1 = \frac{227406.4446}{5748.0788} = \underline{39.5622 \text{ ml/min/mmHg STPD}}$$

$$Vc^1 = \frac{0.57 \times 502.1692 \times 28.8796 \times 15.8855}{100 \times 12.9941}$$

$$Vc^1 = \frac{131315.8061}{1299.41} = \underline{101.0580 \text{ mls.}}$$

$$\frac{Dm^1}{Vc^1} = \underline{0.3915}$$

$$KCO = \frac{TL \text{ (low tension Oxygen)}}{Va \text{ (low tension Oxygen)}} = \underline{4.0701}$$

Note. These results are given to four places of decimals purely because the computer calculated to this precision. It does not mean that the values have anything like that precision.

LUNG FUNCTION RESULTS. COMPUTER PRINT OUT. DATA FOR CALCULATION ON REVERSE SIDE OF SHEET.

DAY 3.0
MONTH 7.0
YEAR 67.00

PATIENT NO. 22.0

WEIGHT KG	TOTAL HT CM	STEM HT CM	BSA SQ M	AGE	
72.0	176.	90.0	1.883	35.	
IC	ERV	FRC	RV	TLC	RV/TLC%
3.778	2.463	4.273	1.810	8.051	22.480
	V INSP	VW	VS	BHT (SEC)	VIT CAP
OXYGEN	5.565	.7867	.6267	8.6705	6.241
AIR	5.125	.7100	.8267	8.7861	
	VA	VA^1	TL	TL^1	
OXYGEN	7.375	7.025	16.690	15.897	
AIR	6.935	7.095	28.216	28.868	
	PAO2	KCO	RV^1	RV^1/RV	
OXYGEN	615.76		1.460	.8064	
AIR	113.6	4.068	1.970	1.089	
	PREDICTED TL^1		TL^1 % OF NORMAL		
AIR	32.480		88.879		
DM	VC	DM/VC	DM^1	VC^1	DM^1/VC^1
36.516	116.94	.31226	39.509	101.26	.39015

DATA TAPE	NO. OF DATA	DATA		VALUE	DATA TEST
-22000000+2+	1	Subject No.		22	
-72000000+2+	2	Weight (Kg.)	W	73·0	
-17600000+3+	3	Standing Height (cm)	H	176	
-90000000+2+	4	Sitting Height (cm)		90·0	
-30000000+1+	5	Day		3	
-70000000+1+	6	Month		7	
-67000000+2+	7	Year		67	
-20000000+2+	8	Temperature	C	20	
-76500000+3+	9	Bar. Press. (mm Hg).	B	765	
-11400000+2+	10	Initial Helium		11·40	
-64500000+1+	11	Final Helium		6·45	
-10290000+2+	12	Insp. Res. Vol. (cm)	IC	10·29	
-67100000+1+	13	Exp. Res. Vol. (cm)	ERV	6·71	
-12840000+2+	14	Final Vol.		12·84	
-12200000+2+	15	Initial Vol.		12·20	
-18000000+1+	16	Oxygen uptake (cm/2min)		1·80	
-10000000+3+	17	HB%		100	
-83000000+1+	18	Back Tension CO (initial)		8·3	
-98400000+2+	19	Back Tension O (initial)		98·4	
-18400000+2+	20	Back Tension Matching CO		18·4	
-90000000+0+	21	Back Tension Matching He		0·90	
-18800000+1+	22	Vol. of Sample (cm)	Vs	1·88	
-23600000+1+	23	Vol. Rejected	Vw	2·36	
-12450000+2+	24	Inspired He	He ins	12·45	
-75000000+1+	25	Breath holding time	bht	7·50	
-00000000+0+	26			0	
-14810000+2+	27	Vol. Inspired	V ins	14·81	
-10710000+2+	28	Expired He	He s	10·71	
-85300000+2+	29	Expired C	Cs	85·3	
-71900000+1+	30	Matching He	He m	7·19	
-70800000+1+	31	Matching He	He m	7·08	
-49700000+2+	32	Matching CO	CO m	49·7	
-49100000+2+	33	Matching CO	CO m	49·1	
-49700000+2+	34	Expired CO	CO ex	49·7	
-35000000+2+	35	Age		35	
-24800000+1+	36	Vol. of sample	Vs	2·48	
-21300000+1+	37	Vol. rejected	Vw	2·13	
-14390000+2+	38	Inspired He	He ins	14·39	
-76000000+1+	39	Breath holding time	bht	7·60	
-00000000+0+	40			0	
-13640000+2+	41	Vol. Inspired	V ins	13·64	
-10080000+2+	42	Expired He	Hes	10·08	
-17200000+2+	43	Expired O	Cs	17·2	
-62200000+1+	44	Matching He	Hem	6·22	
-58800000+1+	45	Matching He	Hem	5·88	
-46100000+2+	46	Matching CO	COm	46·1	
-44000000+2+	47	Matching CO	COm	44·0	
-45400000+2+	48	Expired CO	COex	45·4	
	49				

LUNG FUNCTION RESULTS. COMPUTER PRINT OUT. DATA FOR CALCULATION
ON REVERSE SIDE OF SHEET. (SHORT PROGRAMME EXCLUDING Dm and Vc).

PATIENT NO. 22.

DATE 3.7.67

WT (KGS)	HT (CMS)	STEM HT (CMS)	
72.	176.	90.	
	B.SA. (SO.M.)	AGE	
	1.883	35.	
	FEV 1.0/FVC%	PFR	VIT CAP
ACTUAL	84.790	610.	6.241
PREDICTED	82.434	602.	
	RV/TLC %	TL^1	TL
ACTUAL	22.479	23.867	28.215
PREDICTED	28.023	32.480	
V INSP.	VW	VS	
5.125	.709	.826	
VA	VA^1	RV^1	TL^1 % NORM.
6.935	7.095	1.970	88.879
RV^1/RV %		KCO	
108.855		4.068	
BHT	IC	ERV	
8.786	3.777	2.463	

DATA TAPE	NO. OF DATA	DATA		VALUE	DATA TEST
-22000000+2+	1	Subject No.		22	
-72000000+2+	2	Weight (Kg.)	W	72·0	
-17600000+3+	3	Standing Height (cm)	H	176	
-90000000+2+	4	Sitting Height (cm)		90·0	
-30000000+1+	5	Day		3	
-70000000+1+	6	Month		7	
-67000000+2+	7	Year		67	
-20000000+2+	8	Temperature C		20	
-76500000+3+	9	Bar. Press. (mm Hg)	B	765	
-11400000+2+	10	Initial Helium		11·40	
-64500000+1+	11	Final Helium		6·45	
-10290000+2+	12	Insp. Res. Vol. (cm)	IC	10·29	
-67100000+1+	13	Exp. Res. Vol. (cm)	ERV	6·71	
-12840000+2+	14	Final Vol.		12·84	
-12200000+2+	15	Initial Vol.		12·20	
	16	Oxygen Uptake (cm/2min)			
	17	HB%			
	18	Back Tension CO (initial)			
	19	Back Tension O (initial)			
	20	Back Tension Matching CO			
	21	Back Tension Matching He			
	22	Vol. of Sample (cm)	Vs		
	23	Vol. Rejected	Vw		
	24	Inspired He	He ins		
	25	Breath holding time	bht		
-57200000+1+	26	F.V.C.		5·72	
	27	Vol. Inspired	V ins		
	28	Expired He	He s		
	29	Expired C	Cs		
	30	Matching He	He m		
	31	Matching He	He m		
	32	Matching CO	CO m		
	33	Matching CO	CO m		
	34	Expired CO	CO ex		
-35000000+2+	35	Age		35	
-24800000+1+	36	Vol. of sample	Vs	2·48	
-21300000+1+	37	Vol. rejected	Vw	2·13	
-14390000+2+	38	Inspired He	He ins	14·39	
-76000000+1+	39	Breath holding time	bht	7·60	
-48500000+1+	40	F.E.V. 1,0		4·85	
-13640000+2+	41	Vol. Inspired	V ins	13·64	
-10080000+2+	42	Expired He	Hes	10·08	
	43	Expired O	Cs	—	
-62200000+1+	44	Matching He	Hem	6·22	
-58800000+1+	45	Matching He	Hem	5·88	
-46100000+2+	46	Matching CO	COm	46·1	
-44000000+2+	47	Matching CO	COm	44·0	
-45400000+2+	48	Expired CO	COex	45·4	
-61000000+3+	49	P.F.R.		610	

APPENDIX

Example of Calculation for Exercise and Standardized Ventilation

Name: — Dkyd. No. — Series No. —

Wt. 80.7 Kg. BP 749 mmHg. Date —

Temp 15 °C Step At 9 ins M Rate 19 per min.

Work level 350 kg/min.

ETT	0	½	1	1½	2	2½	3	3½	4	4½	5
Rest [1.]	435	438	441	444	448	452	455				
Exercise [2.]	–	461	468	477	488	501	514	527	541	556	570
Recovery [3.]	–	582	591	599	606	611	617	622	626	630	634
Recovery [4.]	–	638	642	646	650	653	657				

Factor = 1.129

RV 1 BTPS/min.		EV 1 BTPS/min.			EEV 1 BTPS		SV 1 BTPS/min.	
Meter 5(1)	455	Meter 5(2)	570	T = Time of exercise in minutes. ÷3	Meter End Recovery	657	EEV / T	26·24
Meter 0	435	Meter 4(2)	541		Meter 5(1)	455	RV	7·45
Subtract	20	Subtract	29		Subtract	202		
÷ 3	6.6			t = total time of exercise and Recovery in minutes. + 13	x Factor	228		
					RV x t	968		
X Factor	7·45	X Factor	37.75		Subtract	131·2	ADD	33·69

RV = Resting ventilation 1/min.
EV = Exercise ventilation 1/min.
EEV = Excess exercise ventilation 1/min.
SV = Standardized ventilation 1/min.

Factor corrects volumes to litres BTPS.

X Factor indicates that the value should be multiplied by the factor, (obtained from Tables) to convert volume of air to BTPS.

APPENDIX

TABLES OF DATA ON REPRODUCIBILITY OF MEASUREMENTS

Forced Vital Capacity

Subject No.	FVC Expt A	FVC Expt B
1	4.88	5.02
8	3.64	3.66
9	5.23	5.11
10	4.03	4.04
11	4.73	4.70
12	4.51	4.53
13	3.74	3.65
14	4.28	4.31
15	4.48	4.27
16	3.87	3.81

Forced Expiratory Volume (1 second)

Subject No.	$FEV_{1.0}$ Expt A	$FEV_{1.0}$ Expt B
1	3.72	3.77
8	2.63	2.62
9	4.52	4.44
10	3.73	3.74
11	3.67	3.57
12	3.56	3.62
13	3.32	3.39
14	3.64	3.61
15	3.55	3.49
16	3.27	3.25

Lung Volumes. Vital Capacity

Subject No.	VC Expt A	VC Expt B
1	5.01	5.27
2	5.23	4.88
3	4.60	4.30
4	4.17	3.88
5	3.60	3.30
6	4.85	4.85
7	5.99	6.29
8	3.84	4.39
9	5.35	5.27
10	3.64	3.61
11	5.34	5.18
12	4.73	4.63

Residual Volume

Subject No.	RV Expt A	RV Expt B
1	0.82	0.52
2	1.58	1.54
3	1.22	1.25
4	1.62	1.76
5	2.29	2.17
6	1.17	1.09
7	1.47	1.13
8	1.68	1.62
9	0.84	0.83
10	1.88	1.93
11	1.01	1.00
12	0.82	1.02

Total Lung Capacity

Subject No.	TLC Expt A	TLC Expt B
1	5.83	5.79
2	6.82	6.41
3	5.82	5.56
4	5.79	5.64
5	5.89	5.47
6	6.02	5.94
7	7.46	7.42
8	5.51	6.01
9	6.19	6.09
10	5.52	5.54
11	6.35	6.18
12	5.55	5.65

Alveolar Volume. Diffusion (Volume Inspired + Residual Volume). Using O_2 Insp. Gas

Subject No.	Va Expt A	Va Expt B
1	5.50	5.49
2	6.24	6.09
3	5.33	5.37
4	4.94	5.20
5	5.29	5.01
6	5.96	5.78
7	6.66	6.94
8	4.99	4.86
9	5.53	5.68
10	4.74	5.21
11	5.51	5.59
12	5.23	5.38

Alveolar Volume (Using AIR Insp. Gas)

Subject No.	Va Expt A	Va Expt B
1	5.48	5.46
2	6.48	6.26
3	5.29	5.43
4	5.26	5.28
5	5.29	5.32
6	5.89	5.78
7	7.30	6.85
8	4.88	4.79
9	5.68	5.74
10	5.17	5.25
11	5.31	5.47
12	5.22	5.50

Alveolar Breath-holding Volume (10 sec) using O_2 Insp. Gas

Subject No.	Va^1 Expt A	Va^1 Expt B
1	5.06	5.76
2	5.70	5.58
3	5.30	5.25
4	4.83	4.71
5	4.69	4.80
6	6.15	5.62
7	6.54	7.05
8	4.52	4.54
9	5.38	5.73
10	4.00	4.17
11	5.47	5.74
12	5.43	5.37

Alveolar Breath-holding Volume (Using AIR insp. gas)

Subject No.	Va^1 Expt A	Va^1 Expt B
1	5.15	5.81
2	6.59	6.01
3	5.33	5.38
4	5.51	5.14
5	4.86	4.79
6	5.76	5.76
7	7.24	7.29
8	4.56	4.63
9	5.66	5.81
10	4.32	4.40
11	5.49	5.80
12	5.61	5.56

Transfer Factor (Using full alveolar Volumes) High O_2 Tension

Subject No.	T_L Expt A	T_L Expt B
1	11.12	11.97
2	15.64	16.61
3	13.68	12.20
4	8.18	7.71
5	13.61	13.65
6	17.69	17.89
7	15.33	15.26
8	10.43	11.48
9	16.00	14.19
10	9.22	10.45
11	12.09	11.96
12	14.42	15.53

Transfer Factor (Low O_2 Tension)

Subject No.	T_L Expt A	T_L Expt B
1	26.24	25.15
2	32.89	32.56
3	24.78	24.92
4	21.58	19.58
5	34.30	35.46
6	35.98	34.68
7	29.32	28.25
8	25.29	24.51
9	30.96	30.99
10	25.15	24.60
11	28.80	29.62
12	31.00	31.65

Transfer Factor (Using Alveolar Breath-holding Vol.) OXYGEN

Subject No.	T_L^1 Expt A	T_L^1 Expt B
1	10.22	12.57
2	14.29	15.22
3	13.58	11.92
4	8.01	6.99
5	12.08	13.06
6	18.27	17.40
7	15.05	15.49
8	9.44	10.72
9	15.41	14.30
10	7.79	8.35
11	12.00	12.27
12	14.97	15.50

Transfer Factor (Using Va^1) AIR

Subject No.	T_L^1 Expt A	T_L^1 Expt B
1	26.37	26.77
2	33.43	31.25
3	24.97	24.70
4	21.16	19.05
5	31.51	31.94
6	35.18	34.57
7	29.10	30.04
8	23.62	23.73
9	30.84	31.39
10	21.02	20.64
11	29.75	31.38
12	33.34	32.02

Diffusion Capacity of Alveolar Capillary Membrane (Dm)

Subject No.	Dm Expt A	Dm Expt B
1	46.89	41.79
2	51.49	48.37
3	33.01	38.08
4	42.74	35.51
5	64.20	73.09
6	56.64	52.43
7	44.62	39.43
8	49.61	40.56
9	43.75	50.72
10	54.37	45.12
11	51.43	56.01
12	49.90	47.28

Volume of Blood in Alveolar Capillaries (Vc)

Subject	Vc Expt A	Vc Expt B
1	59.13	63.13
2	92.22	102.11
3	86.76	64.31
4	40.78	39.96
5	61.38	57.44
6	88.70	94.61
7	87.50	93.00
8	48.51	58.05
9	96.38	75.64
10	39.63	50.45
11	60.36	59.18
12	79.04	93.01

Standard Ventilation

Subject No.	SV Expt A	SV Expt B
1	24.32	27.39
8	33.77	34.07
9	36.82	35.04
10	35.66	36.93
11	34.30	29.08
12	32.85	32.26
13	32.18	33.85
14	32.34	36.85
15	39.15	39.69
16	29.81	25.96

Exercise Ventilation

Subject No.	EV Expt A	Expt B
1	24.35	30.40
8	33.20	33.10
9	39.20	38.05
10	37.00	36.70
11	33.40	32.40
12	36.65	34.60
13	31.95	32.80
14	33.00	34.10
15	38.35	38.20
16	31.25	25.80

A PROPORTIONAL MORTALITY STUDY OF MALIGNANT NEOPLASMS OF THE LUNG AND GASTRO INTESTINAL TRACT IN PLYMOUTH MALES

PROPORTIONAL MORTALITY STUDY

MALIGNANT NEOPLASMS OF THE LUNG AND GASTRO INTESTINAL TRACT

An association between asbestosis and bronchial carcinoma was reported in 1935 (Lynch and Smith; Gloyne). In reports of the Chief Inspector of Factories for 1947, 1954 and 1955, Merewether drew attention to this association and in 1955 Doll showed that there was a tenfold increase in the risk of developing lung cancer in men with asbestosis who had worked in an asbestos factory for 20 years or more. In recent papers describing the causes of death amongst workers in the same factory (Knox, Doll and Hill, 1965; Hill, Doll and Knox 1966; Knox, Holmes, Doll and Hill 1968), it is shown that men and women employed in scheduled areas after 1933 have had a mortality experience close to the national average. Men employed for 10 years or more before 1933 showed increased mortality for lung cancer and for diseases of the respiratory and circulatory systems associated with asbestosis. Men employed for less than 10 years before 1933 showed an increased mortality for lung cancer, but not for other causes. The asbestos industry regulations 1931 were fully implemented by 1933 and continuous improvement in working conditions has taken place since then. It would be interesting to carry this study further and to include all workers in this factory, not only those in the scheduled areas, as many of the other workers would have had varying degrees of asbestos exposure.

Jacob and Anspach (1965) in a study of male and female asbestos workers in Dresden have shown that while there has been a reduction in the number of cases with right heart failure as the cause of death, there has been an increase in the number of cases with neoplasms of the lung and pleura. They suggest that, because of the lower dust concentrations, men

are now living long enough to develop malignant tumours instead of dying of cor pulmonale as the terminal event of asbestosis.

Braun and Truan (1958), in a study of lung cancer in asbestos miners, review the literature and point out that most other surveys then showed some bias by dealing with selected material. The selection being either because of referral to a specialist unit, or because of autopsy, as well as the difficulties of defining "asbestosis". They present a study of Quebec asbestos miners and suggest that the data shows that there is no increased risk of lung cancer in these men.

The findings of this paper have been criticised by Mancuso (1965) and the most important criticism is that, of the 5,958 men studied, 30% had less than 10 years asbestos exposure and 70% less than 20 years exposure. All but one of the 9 proven or 12 total cases of lung cancer occurred in the men exposed for more than 20 years.

This is valid criticism. It would be very desirable to study the fate of the selected population after another 10 years to see if these findings were confirmed, or whether there was an increase in lung cancer with the increase in length of time from first exposure.

It must be remembered that these men were exposed to chrysotile asbestos and mineral dust only, so it would be very important to confirm this finding, as there is some suggestion that chrysotile asbestos is less harmful than the other varieties.

Mancuso and Coulter (1963) and Selikoff and his colleagues (1964, 1968) similarly report the increased incidence of lung cancer in asbestos workers and also point out that this increase relates to asbestos exposure and is not limited to those having asbestosis. These papers are also

important in that they relate to asbestos insulation workers who handle asbestos products and not to asbestos textile workers. The period of exposure was long, the minimum length of time from first exposure in the men under review being 20 years. Attention is drawn to the excess risk caused by the combination of asbestos exposure and cigarette smoking. This had previously been suggested by Todd et al (1956) Isselbacher et al (1953) and Anderson and Campagna (1960), as being worthy of further study.

It was also suggested in some of these studies that there is an increased risk of gastro intestinal neoplasms in workmen exposed to asbestos. In 1954 Leicher reported a patient with asbestosis and an abdominal tumour, and Bonser et al (1955) drew attention to the high incidence of abdominal tumours in their series of 72 cases of asbestosis. Four of these had abdominal neoplasms. Keal (1960) reported one male death from peritoneal neoplasm in 15 male deaths with asbestosis, and 9 female deaths from ovarian or peritoneal neoplasms in 15 female deaths with asbestosis.

Mancuso and Coulter (1963) found 5 peritoneal neoplasms when 0.08 were expected.

Selikoff, Churg and Hammond (1964) reported 29 cases of cancer of the stomach, colon and rectum when 9.4 were expected and in 1968 the same authors found 8 deaths from cancer of these sites where 1.8 were expected. They point out that the figures are small and they do not wish to draw conclusions from them at this stage.

Doll's 1955 series showed only 3 cases of intra abdominal neoplasms, and his latest report (Knox, Holmes, Doll and Hill 1968) suggests that the deaths of asbestos textile workers due to other neoplasms than lung cancer were not statistically significant. Of the five deaths reported, one was attributed to cancer of the

stomach, two to cancer of the colon, one to cancer of the gall bladder and one to cancer of the prostate.

It would appear that there is agreement that patients with asbestosis have an increased risk of developing lung cancer. Some series suggest that more than 50% of persons with asbestosis will develop this tumour (Buchanan 1965).

American and German workers suggest that there is an increased risk of lung cancer in persons exposed to asbestos, but who do not have asbestosis, and American workers also suggest the possibility of an increased risk of gastro intestinal cancers. Smokers who work with asbestos have a greatly increased risk of developing lung cancer.

British experience (in an asbestos textile factory) suggests that when there was a high incidence of asbestosis, then there was also an increased risk of lung cancer, but that when the incidence of asbestosis was reduced, the risk of lung cancer also fell to the expected rate for the general population.

Studies by Newhouse (1968) from an asbestos factory population, and Elmes (1968) from insulation workers suggest that there is an increase in lung cancer, and possibly gastro intestinal cancers in asbestos exposed workers without necessarily having asbestosis, but these studies are not yet complete.

Mesothelioma of the Peritoneum and Pleura.

Following the report by Wagner, Sleggs and Marchand (1960) of an association between asbestos exposure in crocidolite miners and mesothelial tumours many reports have appeared which support this association. The reports of McCaughey et al (1962) Owen (1964) Selikoff, Churg and Hammond (1964) Elmes and Wade (1965) and Selikoff et al (1967) relate to workers

exposed to asbestos as insulation workers or by working near them. Those of Hourihane (1964) Enticknap and Smither (1964) and Newhouse and Thompson (1965) relate to those working in asbestos factories or, in some cases, living in the neighbourhood of those factories.

It is not yet known whether or not crocidolite is the only form of asbestos which can cause these tumours in man, but the evidence suggests that the risk of developing pleural or peritoneal mesothelioma is higher in men exposed to this rather than the other forms of asbestos.

Most of these reports draw attention to the long period of time from first exposure to asbestos before the development of these tumours, and also to their occurrence many years later, following short and perhaps trivial exposure to asbestos.

Because of these reports we wondered if haphazard exposure to asbestos of large numbers of men in Devonport Dockyard had resulted in an increase in malignant neoplasms of the lung and gastro intestinal tract.

The uses of asbestos and the men exposed to the dust have previously been described, but it is important to remember that although all types of asbestos have been used in the Dockyard since 1880, it is only since 1946 that they have been used in such very large quantities.

The process of spraying crocidolite was undertaken by contractors in 1945, then by Dockyard employees in 1946-47 reaching a peak in 1949-53 and ceasing in 1963. The problem of removing the material remains.

Similarly large amounts of pipe lagging have been applied and removed and here the Dockyard employees were directly involved from 1946 onwards.

From the dust sampling survey it can be seen that for many years large numbers of men have been exposed, often unknowingly and inter-

mittently to varying, but sometimes very high concentrations of asbestos dust.

There were no accurate records of the numbers and ages of men employed in the Dockyard over the last 10 years, and death rates could not be calculated. It was decided to calculate the proportions of deaths for selected causes from the total deaths in Dockyard workers and compare these with the proportions for these causes occurring in the total deaths of other Plymouth males. The method is similar to that described by Bradford Hill and Lewis Faning (1948) for arsenic workers and Doll (1958) for nickel workers.

Methods

Details of all male deaths over the age of 15 years from 1st January 1960 to 31st December 1967 were extracted from the Returns of Deaths to Sanitary Authorities held by the Medical Officer of Health for the City of Plymouth. Similar details were obtained for 1958 and 1959 from death registers of the Plymouth Superintendent Registrar of Deaths. The Medical Officer of Health for Plympton and Plymstock, which became part of Plymouth in 1967, provided the same data for those areas.

Deaths occurring in Plymouth of men who were not normally resident in the City were excluded, but details of deaths occurring outside Plymouth of men normally resident in the area were obtained from the forms of Transferable deaths and included in the data.

The data available for each man included his age, address, occupation and cause of death, and these were coded for transfer onto punch cards. The causes of death had been coded by the staff of the Medical Officer of Health according to the Intermediate List (A) of 150 causes of death from the International Classification of Diseases 1955 (1957). This coding was

used because I had nobody with sufficient experience to apply the full International Classification list. The causes and their International List numbers are given in Table 5.I.

The coded data was sorted into two main occupational groups, Dockyard employees, and all other workers. It was possible to identify Dockyard workers by the invariable addition of the letters H.M.D. after the occupation by the Registrars at the time of death certification. It was a most unlikely occurrence for this not to have been done, but in the few cases in which it was omitted then, of course, the bias would favour the non-Dockyard group, and will not tend to over emphasise any trend in the Dockyard group.

The deaths in each main occupational group were tabulated in 5 year age groups under the causes of death given in Table 5.I. This was done for each year and for the 10 years 1958-67 and the data for the whole period are set out in Table 5.II. The data for individual years are given at the end of this section.

The deaths occurring for each cause in Table 5.I for 1958-67 are presented as percentages of the total deaths in each group in Table 5.III. From Figure 5.1 it will be seen that there are no great differences between the groups, but for lung cancer there is a slight excess in the Dockyard group for nine of the ten years. The differences between the groups for each year are not significant. The average age at death of Dockyard workers is 68.6 years and that of non-Dockyard workers 68.8 years.

The use of direct proportions does not take into account the differences in age structure between the two groups, so that a method of standardising for age is required. The method suggested by Doll (1958) has been used. For each 10 year age group the number of deaths occurring in

Dockyard workers was compared with the expected number of deaths in that group. The latter was obtained by assuming it to be in the same proportion to deaths from diseases other than lung or gastro-intestinal cancer it was in the non-Dockyard group. Thus for lung cancer in age group 55-64 there were 227 deaths in the non-Dockyard group and 1,616 deaths from all other causes apart from lung or gastro-intestinal cancer. In the Dockyard group there are 725 deaths from all causes other than lung or gastro-intestinal cancer so we would expect $\frac{227}{1616} \times 725 = 101.8$ deaths from lung cancer. 114 were observed.

This calculation was made for each age group and the results added to give the total expected values.

The values for observed minus expected numbers of deaths in age groups are given for each of the 10 years 1958-67 in figure 5.2, and the total values for all ages for individual years in figure 5.3.

Although there is again a suggestion that there is an excess of cases of lung cancer in Dockyard workers the differences are not statistically significant.

The value for observed minus expected deaths for lung cancer is more than twice its standard error in only two groups, 1962 ages 75 and over; and 1961 ages 45-54. For gastro-intestinal cancer this occurs in 1961 for ages 75 and over and in 1959 for ages 65-74. For the 10 year totals the excess value is never twice its standard error for lung or gastro-intestinal cancer.

In an Appendix the difficulties of making a comprehensive appraisal of these figures are discussed, and another form of analysis is presented which also confirms that there is no statistically significant increase in lung cancer or gastro-intestinal cancer in Dockyard workers shown in this data. (See Appendix to this section).

FIG 5.1 DEATH FOR EACH CAUSE EXPRESSED AS PERCENTAGES OF THE TOTAL DEATHS IN DOCKYARD WORKERS AND ALL OTHER PLYMOUTH MALES

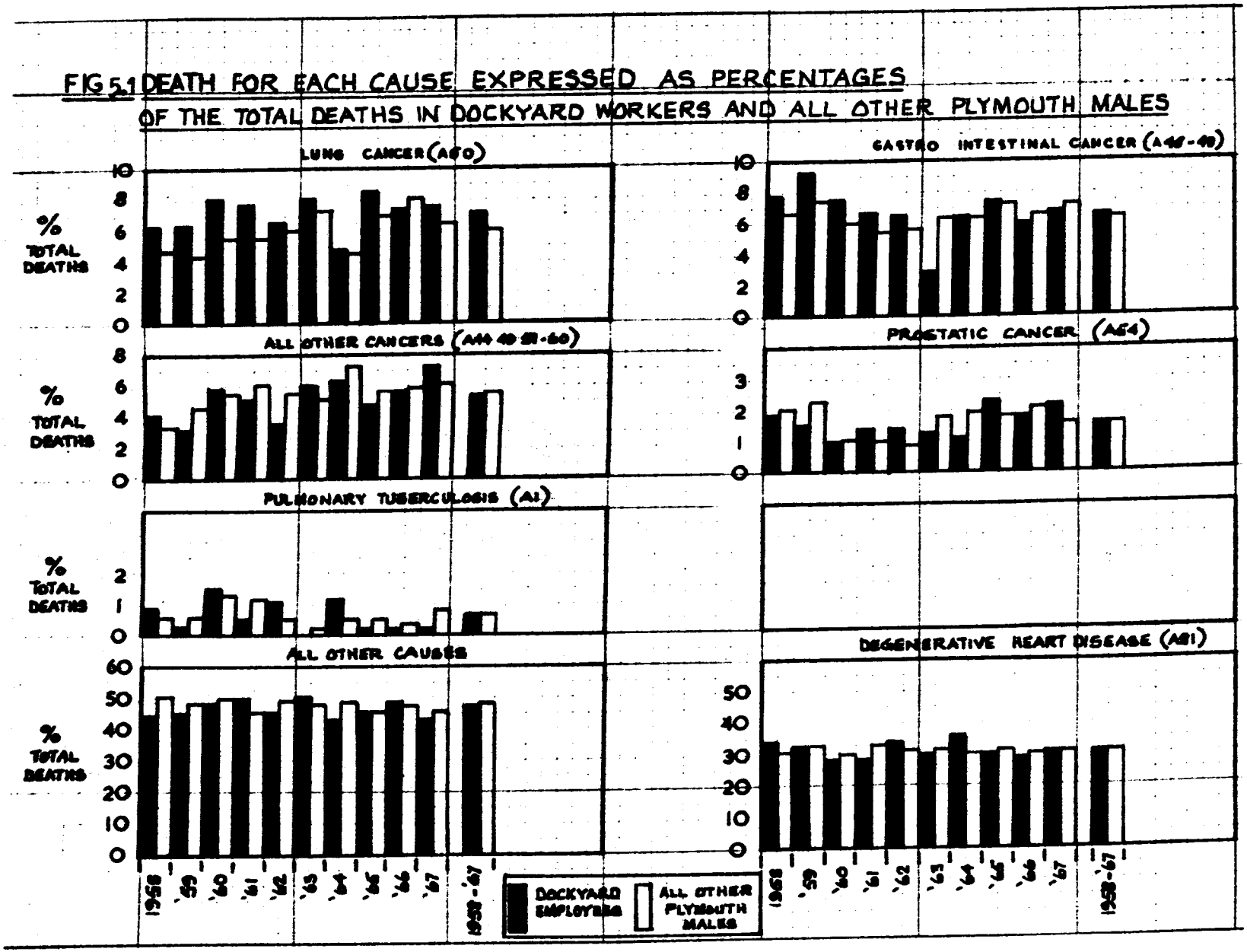


FIG 5.2 DIFFERENCE OF OBSERVED MINUS EXPECTED NUMBERS OF DEATHS FROM LUNG AND GASTRO INTESTINAL CANCER IN DOCKYARD WORKERS. 1958-1967

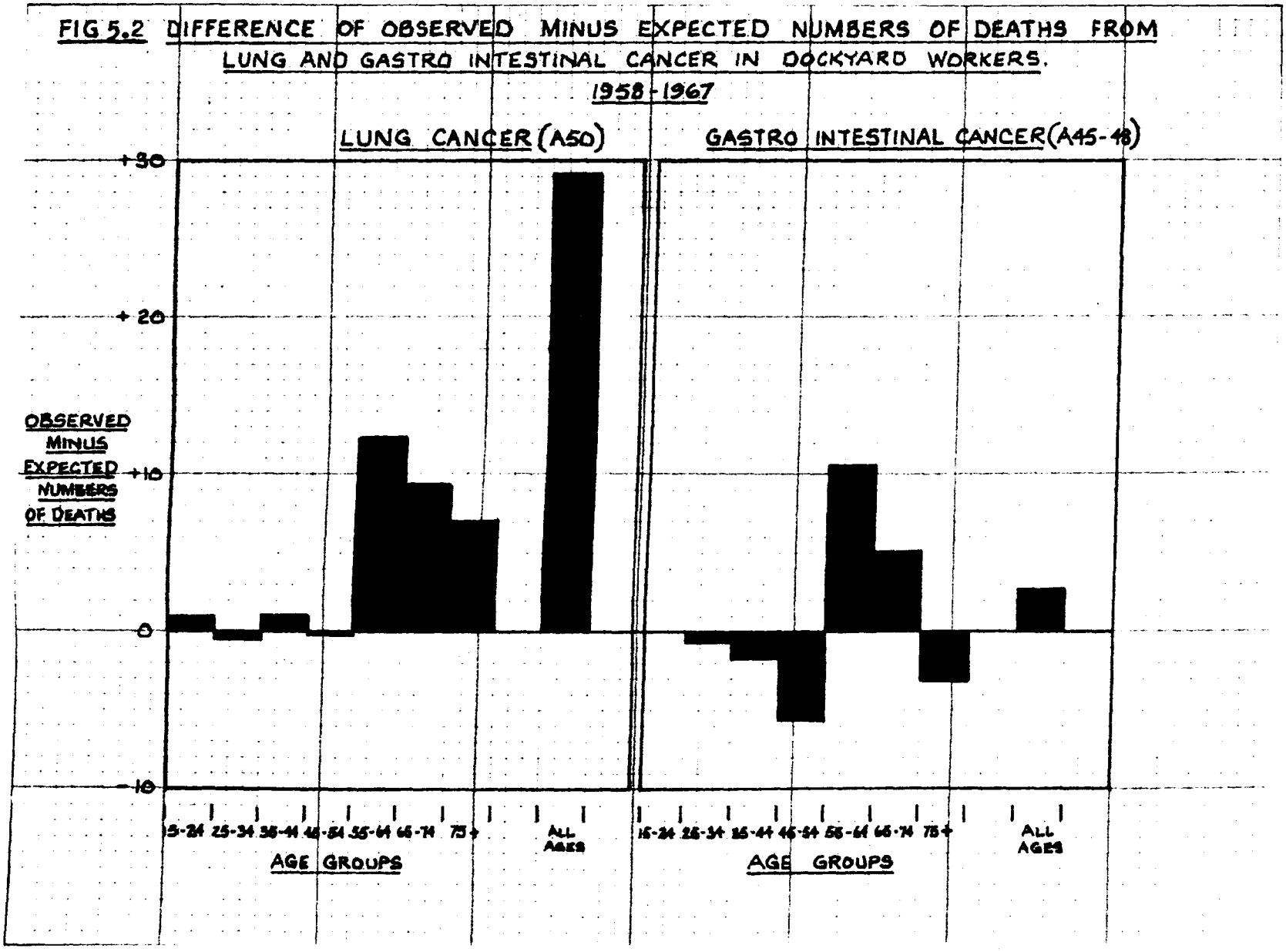
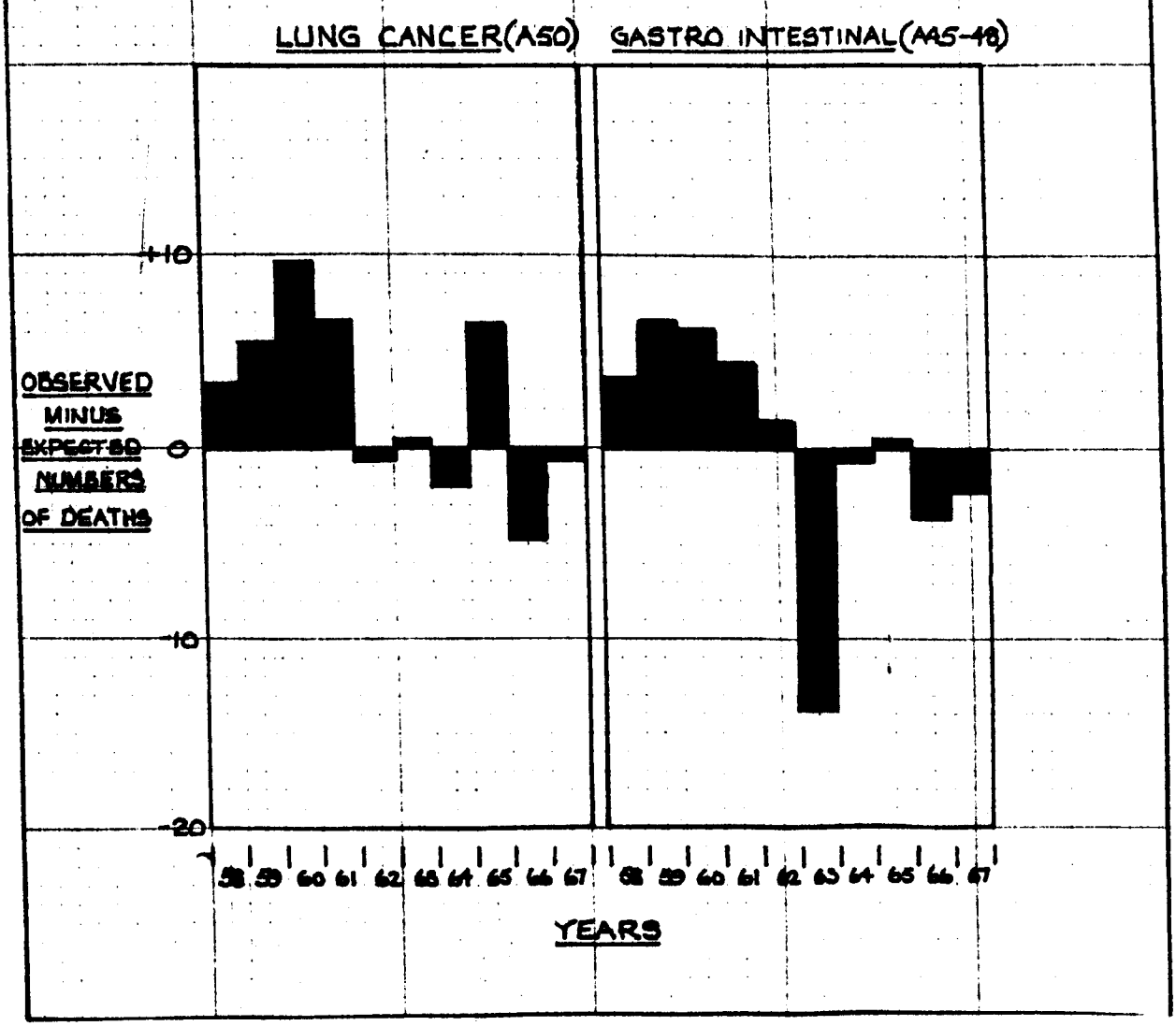


FIG 5.3 DIFFERENCE OF OBSERVED MINUS EXPECTED DEATHS - TOTAL FOR ALL AGES FOR INDIVIDUAL YEARS 1958 - 1967 FROM LUNG AND GASTRO INTESTINAL CANCER IN DOCKYARD WORKERS.



In order to see if we were missing a real increase by considering the population as a whole it was decided to look at various occupational groups who could be expected to have had definite asbestos exposure. It was not possible to identify laggers from the death returns as they are described as skilled labourers along with many other men, so the mortality of laggers is being done as a separate study using Dockyard records. Table 5.IV gives occupational groups likely to have had asbestos exposure.

The numbers of deaths for specific causes expressed as a percentage of total deaths in each group are given in Table 5.V. It can be seen that there is no obvious indication of a significant excess for lung or gastro-intestinal cancers in any group.

Finally it was decided to examine the locality of the man's home to see if men living near the Dockyard died more frequently of lung and gastro-intestinal cancer because of theoretical "environmental or atmospheric" exposure to asbestos. The results are given in Table 5.VI and it can be seen that there is no such suggestion. The only thing to emerge from this study is that a higher percentage of men working in the Dockyard live within one mile of it.

A small number of men working in the Dockyard live on the west side of the Tamar in Saltash, Torpoint and St. German's Rural District Council. Detailed figures were not available for previous years but the data for 1967 showed that there were 24 deaths in Dockyard workers, two of these being gastro-intestinal cancers and one due to lung cancer. There were 108 deaths in all other men in the district and of these 6 were gastro-intestinal cancers and 7 lung cancers.

The figures are very small, and their omission from the main data does not cause any bias.

Discussion

These results show that at present there is no excess of lung or gastro intestinal cancers occurring in Dockyard workers as a whole, or in specific groups of Dockyard workers who might reasonably be expected to have had exposure to higher concentrations of asbestos dust.

It may be that some of the men classified as "other Plymouth males" were at one time Dockyard workers, but the number mis-classified is thought to be very small. The occupations of men with lung cancer in the non-Dockyard group for one year were checked and there were no mis-classifications, but this refers only to the last employment. As a point of interest even the retired Dockyard employees are so described on death certification, e.g. "Shipwright H.M.D. Retired".

The cause of death obtained from death returns leads to some inaccuracies, but these will apply equally to both groups. There are similar sources of error in coding the cause of death, but a check on the coding for two years produced only a few revised codings which made no difference to the results so that it was decided to accept and use the coding applied by the staff of the Medical Officer of Health.

There may in fact be no increase in lung and gastro-intestinal cancers in these men who have been intermittently exposed to asbestos, for two reasons.

Firstly the population contains a large number of men who may have been only very infrequently exposed to asbestos, many who may never have been exposed to asbestos, as well as an undetermined number who have been exposed to varying concentrations. From the dust sampling report it is obvious that many of them have been exposed to high concentrations from time to time, but not continuously. The men continuously heavily exposed form a very

small part of this population. They are the laggers for whom we have incomplete mortality data, and the sprayers. We know the cause of death for 28 of the twenty-nine laggers who are known to have died since 1946. There were five deaths due to malignant disease. One died of lung cancer; the other causes were carcinoma of tonsil; carcinoma of pancreas; carcinoma of urinary bladder; and malignant melanoma with metastases.

Twelve of the 71 men trained as asbestos sprayers are known to have died. Six of these deaths were due to malignant disease. The causes were 1 lung cancer; 1 carcinoma of larynx; 1 carcinoma of prostate; 1 carcinoma of colon; and 2 carcinoma of stomach.

It may be that only those continuously exposed to high concentrations of asbestos are at risk from the development of malignant tumours.

However, we know that men intermittently exposed to asbestos are developing asbestosis and mesothelioma, and we know that a larger number of men are showing signs of asbestos exposure, i.e. calcified and hyaline pleural plaques (Sheers and Templeton 1968). This suggests that if the exposure is sufficient to cause these changes, then it might in time result in an increase in malignant diseases associated with asbestos.

This leads to the second, and perhaps the more likely reason why we have not yet seen an increase in these tumours in Dockyard workers. It is only since 1946 that the really heavy and widespread dispersion of asbestos has occurred. The advent of spraying, and many large refitting tasks since then have meant an enormous increase in the number of men heavily, if intermittently exposed to asbestos.

We know from the studies mentioned previously that there is a long latent period before these tumours occur, and that this is probably thirty years or more. Few of the men in Devonport have been exposed for more

than 20 years, and it is going to be very interesting to see if an increase in these tumours will become apparent over the next 10 years or so. For this reason the present study is important in establishing a base line from which to continue the study.

Arrangements have been made for the study to continue.

TABLE 5.I

CAUSES OF DEATH STUDIED WITH CLASSIFICATION NUMBERS

Cause	Intermediate List A Number	International List Number
Malignant Neoplasm of Prostate	A54	177
Malignant Neoplasm of Gastro Intestinal Tract	A45-48	150-154
Malignant Neoplasm of Trachea Bronchus and Lung not specified as secondary	A50	162-163
All other Neoplasms	A44, 49, 51-60	140-148; 155-160; 164, 165, 170-181; 190-205; 210-239
Arterio sclerotic and degenerative heart disease	A81	420-422
Pulmonary Tuberculosis	A1	001-008
All	Other	Causes

TABLE 5.II

NUMBERS OF DEATHS FROM SPECIFIC CAUSES AMONG MEN IN PLYMOUTH AND DISTRICT SUBDIVIDED BY AGE AND LAST EMPLOYMENT.

1958-67

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	1	1	2	0	7	0	0	0	7	1	0	56	75
20-24	0	0	0	1	0	0	11	0	0	0	12	0	0	63	87
25-29	0	0	0	2	1	0	5	0	0	1	3	1	0	44	57
30-34	0	0	0	2	4	0	10	0	3	2	12	4	1	47	85
35-39	0	1	2	5	2	0	14	1	2	3	13	22	2	55	122
40-44	0	2	2	11	20	3	20	0	14	9	24	51	4	85	245
45-49	0	2	12	3	36	2	29	0	21	35	26	123	3	141	433
50-54	0	7	17	10	44	1	79	3	34	67	34	168	16	201	681
55-59	0	40	52	28	111	3	134	10	67	91	62	283	9	314	1,204
60-64	7	39	62	24	179	5	234	6	85	136	70	388	8	466	1,709
65-69	9	57	63	37	197	2	294	22	100	124	104	417	9	646	2,081
70-74	12	42	38	32	217	4	335	37	114	85	75	514	7	764	2,276
75 +	35	71	32	50	415	4	658	94	243	82	140	1,370	6	2,201	5,401
All Ages Over 15	63	261	281	206	1,228	24	1,830	173	683	635	582	3,342	65	5,083	14,456

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

TABLE 5.III

NUMBER OF DEATHS FOR EACH CAUSE EXPRESSED AS PERCENTAGES OF THE TOTAL DEATHS IN EACH GROUP

1958-1967

	DOCKYARD WORKERS TOTAL NUMBER OF DEATHS = 3,893		NON-DOCKYARD WORKERS TOTAL NUMBER OF DEATHS = 10,563	
	No. of Deaths	% of Total Deaths	No. of Deaths	% of Total Deaths
Prostatic Cancer A54	63	1.6%	173	1.6%
Gastro Intestinal Cancer (A45-48)	261	6.7%	683	6.5%
Lung Cancer (A50)	281	7.2%	635	6.0%
All Other Cancers (A44, 49, 51-60)	206	5.3%	582	5.5%
Degenerative Heart Disease (A81)	1,228	31.5%	3,342	31.6%
Pulmonary Tuberculosis (A1)	24	0.62%	65	0.62%
All Other Causes	1,830	47.0%	5,083	48.1%

TABLE 5.IV

DOCKYARD WORKERS CLASSIFICATION OF OCCUPATIONAL GROUPS

Group	Occupation
I	Electrical Fitter Riveter Welder Burner Driller Caulker Shipfitter Plumber
II	Shipwright Engine Fitter Boilermaker
III	Painter Skilled Labourer Labourer
IV	All Other Occupations

TABLE 5.V

NUMBER OF DEATHS FOR EACH CAUSE EXPRESSED AS PERCENTAGES OF THE TOTAL DEATHS IN EACH OCCUPATIONAL GROUP OF DOCKYARD EMPLOYEES (NUMBERS OF DEATHS IN BRACKETS)

Causes	Occupational Groups				All Dockyard Employees
	I	II	III	IV	
Prostatic Cancer (A54)	1.4% (8)	1.4% (11)	1.8% (30)	1.6% (14)	1.6% (63)
Gastro Intestinal Cancer (A45-48)	5.8% (34)	6.5% (52)	7.1% (117)	6.7% (58)	6.7% (261)
Lung Cancer (A50)	6.8% (40)	8.0% (64)	7.7% (127)	5.8% (50)	7.2% (281)
All Other Cancers (A44, 49, 51-60)	4.1% (24)	5.0% (40)	5.6% (93)	5.7% (49)	5.3% (206)
Degenerative Heart Disease (A81)	30.3% (178)	33.0% (263)	30.3% (499)	33.4% (288)	31.5% (1,228)
Pulmonary Tuberculosis (A1)	0.8% (5)	0.4% (3)	0.9% (15)	0.1% (1)	0.6% (24)
All Other Causes	50.8% (298)	45.7% (364)	46.5% (767)	46.6% (401)	47.0% (1,830)
Total Number of Deaths	587	797	1,648	861	3,893

TABLE 5.VI

PROPORTIONAL MORTALITY ANALYSED FOR ADDRESS

1958-1967

DOCKYARD WORKERS TOTALS		Prostatic Cancer (A54)	Gastro Intestinal Cancer (A45-48)	Lung Cancer (A50)	All Other Cancers (A44, 49, 51-60)	Degenerative Heart Disease (A81)
18.6% (723)	Address within 1 Mile of Dockyard	1.9% (14)	6.4% (46)	8.2% (59)	6.1% (44)	27.5% (199)
81.4% 3,893 (3,170)	All Other Addresses	1.3% (49)	6.8% (215)	7.0% (222)	5.1% (162)	32.5% (1,029)
NON-DOCKYARD WORKERS TOTALS		A54	A45-48	A50	A44, 49, 51-60	A81
6.5%	Address within 1 Mile of Dockyard	1.8% (12)	6.3% (43)	5.0% (34)	5.6% (38)	28.4% (194)
93.5% 10,563 (9,880)	All Other Addresses	1.6% (161)	6.5% (640)	6.1% (601)	5.5% (544)	31.9% (3,148)

APPENDIX

The reasons for using proportional mortality in this study have been explained. This form of analysis has the drawback that there is no obviously appropriate means of accumulating the evidence from different age groups and different calendar years, each of which gives an excess or deficiency of observed deaths compared with those expected. Moreover there are several different scales on which this excess or deficiency can be measured.

Consider the general form of 2 x 2 table given by each year and age group:

	Lung cancer deaths	All other deaths	Total
Dockyard workers	a	b	a+b
Other workers	c	d	c+d
Total	a+c	b+d	a+b+c+d

Doll (1958) calculated the expected number of deaths to be compared with the observed number a by postulating that it should be in the same ratio to b as c is to d, that is,

$$\text{expected number} = \frac{c}{d} \times b$$

and excess of observed over expected is

$$a - \frac{c}{d} \times b$$

$$= \frac{ad - bc}{d}$$

Hill and Faning (1948) postulated that the expected value of a should be the same fraction of a + b as c is of c + d, that is,

$$\text{expected number} = \frac{c}{c+d} \times (a+b)$$

and excess of observed over expected is

$$a - \frac{c}{c+d} \times (a + b)$$

$$= \frac{ad - bc}{c+d}$$

These two measures of excess mortality are not the same, though both are fractions of ad - bc. Yet another measure is given by calculating the expected number as in calculating χ^2 for the 2 x 2 Table. Here it is assumed that the expected number will be the same fraction of a + b as a + c is of the total a+b+c+d. That is,

$$\text{expected number} = \frac{a+c}{a+b+c+d} \times (a+b) \text{ and the excess is}$$

$$a - \frac{a+c}{a+b+c+d} \times (a+b)$$

$$= \frac{ad-bc}{a+b+c+d}$$

Again, the answer is different, but again it is a fraction of ad-bc.

It is not obvious which of these measures is the appropriate one to accumulate over age groups and calendar years. Each gives a weighted sum of the excess deaths, the weights differing. One arbitrary solution is to express each value of ad-bc as a multiple of its standard error, which is

$$\sqrt{\ (a+b)\ (c+d)\ (a+c)\ (b+d)/(a+b+c+d)\ }$$

and so arrange that each excess is weighted inversely as its accuracy. The relevant data are given in Appendix Table 5.I, and the resulting weighted values of ad-bc are given in Appendix Table 5.II.

If there is no excess mortality for lung cancer the resulting numbers in

Table 5.II all constitute a sample of 50 from a Normal distribution with zero mean and unit standard deviation.

The histogram, and the theoretical distribution is shown in Figure 5.1 and it is a fairly good fit.

These numbers are now tested to see if their variability can in part be attributed to consistent differences from one age group to another, and from one year to another, or to differences between age groups which are changing from year to year.

	Degrees of Freedom	χ^2	Significance
Variation from year to year	9	6.048	$0.70<P<0.80$
Variation from age to age	6	3.597	$0.70<P<0.80$
Variation of age effects with year effects	34	36.215	$0.50>P>0.30$
Deviation of average excess from zero	1	1.004	$P\simeq 0.316$

Nothing approaches a high level of significance, and we therefore have no evidence against the supposition that there is no excess of lung cancer mortality in Dockyard workers. This negative conclusion is reinforced by Table 5.III, which gives the estimated average excesses for each of the ten years, allowing for constant effects of the varying age distribution. If anything, the excess has been diminishing over the 10 years studied. The units are still Normal deviates.

Table 5.IV shows the corresponding estimates for the different age

groups, allowing for constant year effects. There seems to be a slight increase of the excess deaths with age, though this is not significant ($P \simeq 0.20$), but even if it were significant it would not be very cogent evidence for an occupational cause since it is in fact a change from a deficiency of cancer deaths in younger ages to an excess in older ages.

I am indebted to Mr. P.D. Oldham for suggesting and carrying out the analysis in the Appendix.

APPENDIX TABLE 5.I

Deaths from lung cancer (A50)

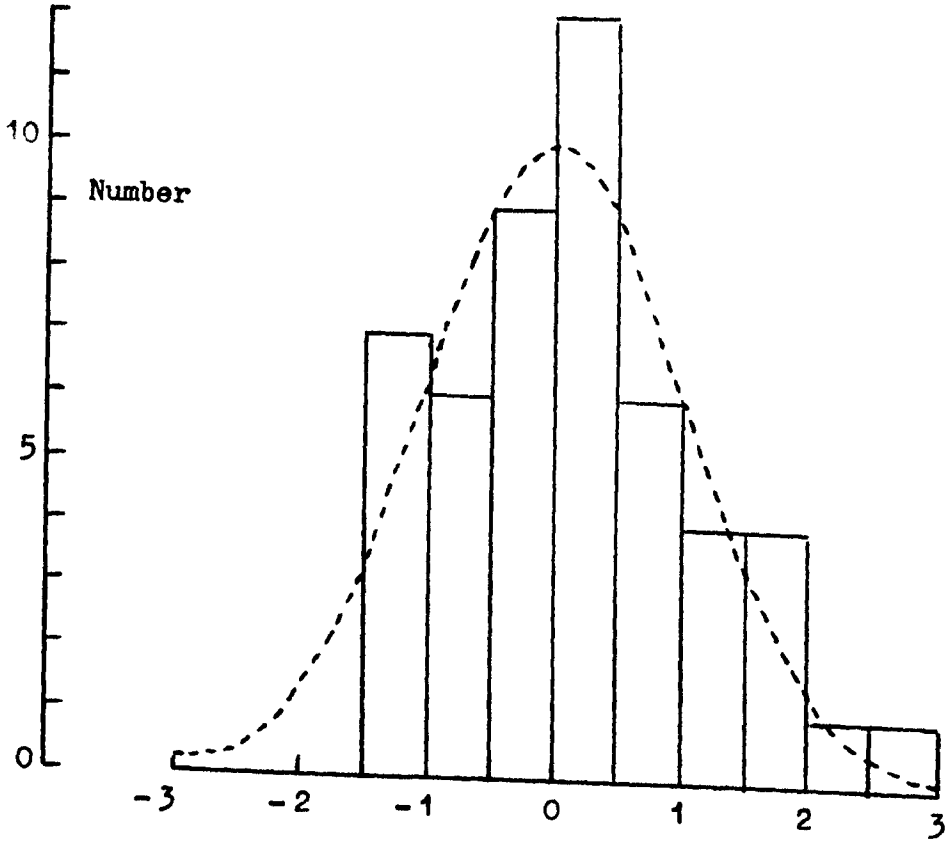
In each cell the entries are ad-bc:a+b+c+d:c+d:d, where the symbols are as in the 2 x 2 table:

	Lung Cancer Deaths	Irrelevant Deaths	Total
Dockyard men	a	b	a+b
Others	c	d	c+d
Total	a+c	b+d	a+b+c+d

Age Group

	15-	25-	35-	45-	55-	65-	75+
1958	0/6/6/6	-2/18/16/15	-4/31/27/26	-134/115/89/77	6/246/180/161	1400/380/284/269	23/503/407/403
59	11/16/11/11	0/9/6/6	0/28/22/22	56/98/81/70	-241/232/161/144	979/348/253/242	458/444/358/355
Year 60	0/18/16/16	0/17/14/14	28/52/40/39	-202/128/106/92	1428/294/202/178	898/409/295/277	-6/528/410/403
61	0/14/12/12	0/20/12/12	15/42/33/31	369/109/90/81	740/265/176/156	-961/418/305/283	120/560/456/444
62	0/13/12/12	0/12/10/10	-18/23/17/14	-166/120/94/84	-677/293/205/181	333/378/279/257	2079/527/412/405
63	0/23/19/19	-1/17/16/15	-22/46/35/33	101/97/73/62	-216/311/227/192	282/443/321/297	-114/570/444/429
64	0/14/12/12	0/13/11/11	0/42/36/36	24/96/72/64	268/279/182/166	-1018/397/261/242	81/506/373/368
65	0/17/14/14	0/15/15/14	14/28/14/13	12/92/68/60	-468/248/172/150	1588/454/301/274	864/498/360/348
66	0/21/19/19	0/7/5/5	-6/19/16/14	-94/104/75/64	40/265/181/147	-548/425/254/230	-265/524/357/349
67	0/20/18/18	0/11/10/10	0/37/29/29	-38/91/69/61	1139/249/158/141	-1297/392/251/224	-683/427/316/307

APPENDIX FIGURE 5.1



APPENDIX TABLE 5.II

Excess lung cancer deaths as multiples of their standard errors.

	Age group						
	15-	25-	35-	45-	55-	65-	75+
1958		- 0.3638	- 0.3913	- 0.7944	+ 0.0114	+ 1.7544	+ 0.0523
59	+ 1.5913			+ 0.4356	- 0.4952	+ 1.4900	+ 1.1739
60			+ 0.9216	- 1.1495	+ 1.7479	+ 0.9588	- 0.0092
Year 61			+ 0.5215	+ 2.4810	+ 1.0970	- 1.0300	+ 0.1442
62			- 1.1035	- 1.0623	- 0.9574	+ 0.4452	+ 2.5873
63		- 0.2577	- 0.8106	+ 0.6601	- 0.2476	+ 0.2543	- 0.1125
64				+ 0.1850	+ 0.4154	- 1.1164	+ 0.1384
65			+ 0.6110	+ 0.0955	- 0.8036	+ 1.1509	+ 0.9068
66			- 0.6474	- 0.5791	+ 0.0509	- 0.4470	- 0.3307
67				- 0.3269	+ 1.7531	- 1.1910	- 1.1670

APPENDIX TABLE 5.III

Estimates of the excess deaths per year, standard error units

Year	Excess
1958	0.1896 ± 0.4389
59	0.6630 ± 0.4707
60	0.5807 ± 0.4878
61	0.7295 ± 0.4878
62	0.0686 ± 0.4878
63	0.0591 ± 0.4389
64	-0.0824 ± 0.5413
65	0.4789 ± 0.4878
66	-0.3039 ± 0.4878
67	-0.2210 ± 0.5413
Average	0.2162 ± 0.2158

APPENDIX TABLE 5.IV

Estimates of the excess deaths
by age group, standard error units

Age group	Excess
15-	1.0851 ± 1.1068
25-	- 0.2189 ± 0.7630
35-	- 0.1697 ± 0.3919
45-	- 0.0055 ± 0.3162
55-	0.2572 ± 0.3162
65-	0.2269 ± 0.3162
75+	0.3384 ± 0.3162
Average	0.2162 ± 0.2158

APPENDIX

TABLES OF CAUSES OF DEATH OF PLYMOUTH MALES 1958-67

APPENDIX

1958

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	0	0	0	0	0	0	0	0	0	0	3	3
20-24	0	0	0	0	0	0	0	0	0	0	1	0	0	2	3
25-29	0	0	0	0	0	0	2	0	0	1	0	1	0	6	10
30-34	0	0	0	0	0	0	0	0	0	0	2	0	1	5	8
35-39	0	0	0	1	0	0	1	0	0	1	0	0	0	8	11
40-44	0	0	0	0	1	0	1	0	1	0	1	5	1	11	21
45-49	0	1	0	0	4	1	4	0	4	7	3	7	0	17	48
50-54	0	1	2	0	9	0	6	1	3	5	1	27	4	17	76
55-59	0	4	4	4	9	0	13	3	6	5	3	31	1	29	112
60-64	0	2	3	0	17	2	14	0	10	14	3	37	0	54	156
65-69	2	3	6	3	16	0	18	1	10	10	11	44	0	59	183
70-74	1	9	4	1	17	0	28	3	12	5	4	60	0	87	231
75 +	3	5	1	4	34	0	54	15	26	4	7	132	0	249	534
All Ages Over 15	6	25	20	13	107	3	141	23	72	52	36	344	7	547	1,396

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1959

	NUMBERS OF DEATHS OF DOCKYARD WORKERS							NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	1	0	0	0	0	0	0	0	2	1	0	2	6
20-24	0	0	0	0	0	0	4	0	0	0	1	0	0	5	10
25-29	0	0	0	0	1	0	0	0	0	0	1	0	0	3	5
30-34	0	0	0	1	0	0	1	0	0	0	0	0	0	2	4
35-39	0	0	0	0	0	0	3	0	0	0	1	3	1	6	14
40-44	0	0	0	2	1	0	0	0	0	0	3	1	0	7	14
45-49	0	0	1	1	3	0	2	0	2	3	1	16	0	13	42
50-54	0	0	2	0	4	0	4	0	2	8	2	21	1	16	60
55-59	0	5	3	2	7	0	17	1	9	8	3	24	1	38	118
60-64	0	4	3	0	16	0	23	0	8	9	4	35	2	36	140
65-69	0	8	3	1	16	0	21	2	8	6	8	44	0	63	180
70-74	3	5	5	2	15	1	28	4	7	5	7	54	1	59	196
75 +	2	7	2	1	42	0	39	15	36	3	12	119	0	209	487
All Ages Over 15	5	29	20	10	105	1	142	22	72	42	45	318	6	459	1,276

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1960

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	-	-	-	-	-	-	1	-	-	-	-	-	-	4	5
20-24	-	-	-	-	-	-	1	-	-	-	1	-	-	11	13
25-29	-	-	-	1	-	-	-	-	-	-	2	-	-	4	7
30-34	-	-	-	-	-	-	2	-	-	-	1	1	-	6	10
35-39	-	-	-	1	-	-	4	1	-	1	5	2	-	7	21
40-44	-	-	1	1	2	1	2	-	-	-	3	4	-	17	31
45-49	-	-	-	-	2	-	3	-	2	4	1	15	1	16	44
50-54	-	2	1	-	6	1	9	1	5	10	7	21	2	28	93
55-59	-	8	10	6	14	2	14	-	8	12	8	28	2	29	141
60-64	2	5	8	1	13	1	21	-	13	12	6	49	2	54	187
65-69	-	6	5	6	17	-	21	4	6	11	9	36	-	65	186
70-74	-	5	5	3	17	-	40	3	14	7	3	55	1	101	254
75 +	2	4	2	4	39	1	70	4	21	7	17	140	2	240	553
All Ages Over 15	4	30	32	23	110	6	188	13	69	64	63	351	10	582	1,545

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1961

	NUMBERS OF DEATHS OF DOCKYARD WORKERS							NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	-	-	-	-	-	-	-	-	-	-	1	-	-	5	6
20-24	-	-	-	-	-	-	2	-	-	-	2	-	-	4	8
25-29	-	-	-	-	-	-	1	-	-	-	-	-	-	7	8
30-34	-	-	-	1	2	-	4	-	-	-	-	-	-	5	12
35-39	-	-	1	-	-	-	1	-	-	1	-	6	1	7	17
40-44	-	-	-	1	1	2	3	-	4	1	1	10	1	5	29
45-49	-	-	5	-	2	-	4	-	3	1	4	12	-	11	42
50-54	-	2	1	2	1	-	4	1	1	8	6	18	3	26	73
55-59	-	2	5	4	11	-	14	1	7	6	6	35	1	31	123
60-64	-	2	9	1	15	-	31	-	4	13	7	32	3	40	157
65-69	2	5	2	3	20	-	27	2	12	14	9	47	3	70	216
70-74	-	2	3	2	24	-	30	-	11	8	13	48	-	91	232
75 +	3	12	3	5	26	-	67	8	21	12	21	181	2	232	593
All Ages Over 15	5	25	29	19	102	2	188	12	63	64	70	389	14	534	1,516

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1962

	NUMBERS OF DEATHS OF DOCKYARD WORKERS							NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	-	-	-	-	-	-	-	-	-	-	-	-	-	8	8
20-24	-	-	-	-	-	-	1	-	-	-	-	-	-	4	5
25-29	-	-	-	-	-	-	-	-	-	-	-	-	-	2	2
30-34	-	-	-	-	1	-	1	-	-	-	1	1	-	6	10
35-39	-	-	-	2	-	-	-	-	-	-	-	1	-	3	6
40-44	-	-	-	-	4	-	-	-	-	3	3	3	-	4	17
45-49	-	-	-	-	6	-	4	-	-	4	2	15	-	20	51
50-54	-	1	1	2	5	-	8	-	3	6	4	19	2	22	73
55-59	-	4	4	1	11	-	17	-	7	9	6	31	1	38	129
60-64	1	3	3	2	20	1	28	1	10	15	13	35	-	56	188
65-69	1	5	7	3	16	1	21	1	7	14	10	38	1	69	194
70-74	-	7	2	-	17	1	30	5	13	8	9	53	1	70	216
75 +	3	3	7	3	44	1	57	3	22	7	13	149	-	240	552
All Ages Over 15	5	23	24	13	124	4	167	10	62	66	61	345	5	542	1,451

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1963

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	0	0	0	2	0	0	0	0	0	0	6	8
20-24	0	0	0	0	0	0	2	0	0	0	2	0	0	11	15
25-29	0	0	0	0	0	0	1	0	0	0	0	0	0	7	8
30-34	0	0	0	0	0	0	0	0	3	1	2	1	0	5	12
35-39	0	0	0	0	0	0	0	0	1	0	0	4	0	3	8
40-44	0	1	0	2	1	0	8	0	3	2	2	8	0	16	43
45-49	0	0	2	0	1	0	3	0	1	6	5	8	0	11	37
50-54	0	0	3	0	4	0	11	0	4	5	3	11	0	24	65
55-59	0	0	6	2	12	0	12	0	5	12	12	34	1	34	130
60-64	0	2	6	4	13	0	29	3	7	23	8	46	0	54	195
65-69	0	1	6	4	17	0	26	3	13	15	11	42	1	74	213
70-74	0	2	4	5	27	0	33	7	16	9	4	58	1	96	262
75 +	5	5	4	6	41	0	70	8	22	15	13	174	0	234	597
All Ages Over 15	5	11	31	23	116	-	197	21	75	88	62	386	3	575	1,593

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1964

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	1	0	0	1	0	0	0	0	0	0	7	9
20-24	0	0	0	0	0	0	0	0	0	0	2	0	0	3	5
25-29	0	0	0	1	0	0	0	0	0	0	0	0	0	5	6
30-34	0	0	0	0	0	0	1	0	0	0	1	0	0	5	7
35-39	0	1	0	0	0	0	0	0	1	0	2	2	0	9	15
40-44	0	1	0	1	3	0	2	0	1	0	7	6	0	10	31
45-49	0	0	2	1	5	1	1	0	0	1	7	11	0	13	42
50-54	0	0	1	2	6	0	5	0	3	7	1	14	0	18	57
55-59	0	6	4	3	14	1	14	2	6	7	8	26	0	28	119
60-64	2	2	6	6	28	1	18	1	10	9	10	45	1	45	184
65-69	1	7	5	6	21	1	37	3	12	11	13	37	0	58	212
70-74	1	3	1	0	27	0	36	3	8	8	8	52	3	65	215
75 +	1	8	2	6	52	1	71	10	23	5	14	119	1	224	537
All Ages Over 15	5	28	21	27	156	5	186	19	64	48	73	312	5	490	1,439

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1965

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	0	0	0	2	0	0	0	1	0	0	7	10
20-24	0	0	0	1	0	0	0	0	0	0	0	0	0	6	7
25-29	0	0	0	0	0	0	0	0	0	0	0	0	0	5	5
30-34	0	0	0	0	0	0	0	0	0	1	2	1	0	6	10
35-39	0	0	1	0	1	0	3	0	0	0	0	0	0	3	8
40-44	0	0	1	1	6	0	1	0	0	1	2	4	0	4	20
45-49	0	0	2	0	3	0	6	0	3	3	1	12	0	9	39
50-54	0	0	1	2	2	0	8	0	6	5	3	13	3	19	62
55-59	0	8	3	1	10	0	16	1	7	7	6	21	1	33	114
60-64	2	6	4	1	22	0	17	0	11	15	4	42	0	42	166
65-69	1	6	14	2	24	0	38	4	15	15	15	50	0	72	256
70-74	3	4	5	7	22	1	36	2	12	12	13	42	0	76	235
75 +	4	9	7	6	45	0	76	11	21	12	11	135	1	190	528
All Ages Over 15	10	33	38	21	135	1	203	18	75	71	58	320	5	472	1,460

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1966

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	0	2	0	0	0	0	0	2	0	0	8	12
20-24	0	0	0	0	0	0	0	0	0	0	2	0	0	7	9
25-29	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2
30-34	0	0	0	0	1	0	1	0	0	0	2	0	0	1	5
35-39	0	0	0	0	0	0	1	0	0	0	2	2	0	3	8
40-44	0	0	0	1	0	0	1	0	1	2	1	3	0	3	12
45-49	0	0	0	1	5	0	1	0	3	5	0	11	1	19	46
50-54	0	1	3	1	5	0	13	0	3	6	4	14	1	14	65
55-59	0	0	6	3	11	0	9	1	8	18	5	19	1	32	113
60-64	0	8	10	2	15	0	28	1	6	16	8	39	0	41	174
65-69	1	6	9	5	27	0	52	1	7	15	6	35	1	58	223
70-74	0	2	5	8	23	1	40	7	9	9	7	50	0	65	226
75 +	8	12	3	7	56	0	93	10	27	8	18	117	0	204	563
All Ages Over 15	9	29	36	28	145	1	239	20	64	79	57	290	4	457	1,458

A54	Prostatic Cancer	A44, 49, 51-60	All Other Cancers
A45-48	Gastro Intestinal Cancer	A81	Degenerative Heart Disease
A50	Lung Cancer	A1	Pulmonary Tuberculosis

APPENDIX

1967

NUMBERS OF DEATHS OF DOCKYARD WORKERS								NUMBERS OF DEATHS OF ALL OTHER PLYMOUTH MALES							TOTAL
Age	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	A54	A45-48	A50	A 44,49, 51-60	A81	A1	All Other Causes	All Causes
15-19	0	0	0	0	0	0	1	0	0	0	1	0	0	6	8
20-24	0	0	0	0	0	0	1	0	0	0	1	0	0	10	12
25-29	0	0	0	0	0	0	1	0	0	0	0	0	0	3	4
30-34	0	0	0	0	0	0	0	0	0	0	1	0	0	6	7
35-39	0	0	0	1	1	0	1	0	0	0	3	2	0	6	14
40-44	0	0	0	2	1	0	2	0	4	0	1	7	2	8	27
45-49	0	1	0	0	5	0	1	0	3	1	2	16	1	12	42
50-54	0	0	2	1	2	0	11	0	4	7	3	10	0	17	57
55-59	0	3	7	2	12	0	8	1	4	7	5	34	0	22	105
60-64	0	5	10	7	20	0	25	0	6	10	7	28	0	44	162
65-69	1	10	6	4	23	0	33	1	10	13	12	44	3	58	218
70-74	4	3	4	4	28	0	34	3	12	14	7	42	0	54	209
75 +	4	6	1	8	36	1	61	10	24	9	14	104	0	179	457
All Ages Over 15	9	28	30	29	128	1	179	15	67	61	57	287	6	425	1,322

A54	Prostatic Cancer
A45-48	Gastro Intestinal Cancer
A50	Lung Cancer
A44, 49, 51-60	All Other Cancers
A81	Degenerative Heart Disease
A1	Pulmonary Tuberculosis

DISCUSSION AND CONCLUSIONS

DISCUSSION AND CONCLUSIONS

MINISTRY OF LABOUR AND NATIONAL SERVICE

Factory Department

St. James Square

London SW1

August 1945.

Dear Sir,

Asbestos Insulation Aboard Ships

I am concerned by the considerable development during the war years in the use of asbestos, either alone or as part of a mixture, in the Shipbuilding and Shiprepairing Industries mainly for the purpose of heat and sound insulation, and the accompanying increase in the number of workers exposed to risks of injury to health through asbestosis.

If the risk is found to continue, the question will arise of including some statutory requirements on the subject when the Factories Act Regulations for constructional and other work in ships are being revised and extended, or of issuing a further code of regulations dealing particularly with the use of asbestos. I would, however, emphasize that, while asbestos dust may not have any apparent effects at first, experience shows that, particularly if the workers are exposed to the dust in substantial concentrations, serious results are apt to develop later. It is therefore important that, even if the work will only be temporary, all reasonably practicable steps should be taken to reduce the risk to a minimum.

I suggest that protection can be secured on the following basis:

(1) In some cases preparatory work, e.g. the making up of insulation mattresses, to which the Asbestos Industry Regulations, 1931,

apply is carried on in a shed in the shipbuilding yard or near the dockside; in such cases those Regulations should, so far as the provisions are applicable, be carefully observed in practice.

(2) On board ship steps should be taken, in accordance with the spirit of the Regulations, to prevent unnecessary concentrations or accumulations of asbestos dust - for instance by having good ventilation arrangements in confined spaces, damping down dust, and clearing up asbestos debris and accumulations of dust as soon as practicable.

(3) The provision of a respirator (Home Office Mark No. 584042 or other approved type) for each workman engaged in the fitting or removal of any dry insulating material containing asbestos, on board ship.

(4) The provision of a similar respirator for all persons engaged in the spraying of asbestos or asbestos mixture and work ancillary thereto which renders them liable to exposure to dust or spray. During spraying, no other person should work in the same compartment unless also provided with a respirator.

(5) No person under 18 should be employed in any process giving rise to asbestos dust or in any compartment or enclosed space where such a process is being carried on.

(6) Specific arrangements for supervising the maintenance, care and use of respirators.

I may say that these arrangements have been accepted by the Shipbuilding Employers' Federation and by the Trade Unions concerned, and I therefore

hope you will be prepared at once to accept the precautions suggested.

I am, Sir,

Your obedient Servant,

(Sgd.) A.W. Garrett

HM Chief Inspector of Factories

The text of this letter to the Shipbuilding and Shiprepairing Industry is given in full because if the suggestions made in it had been fully implemented, then many men might have been prevented from developing diseases associated with asbestos.

Review of the literature has shown that, although some workers (Fleischer 1946, Frost et al 1956, Walters 1959, Leathart 1962, Marr 1964) suspected the presence of the hazard presented by the use of asbestos in shipyards, it was not until fairly recently that evidence has been produced relating to the numbers of men affected. The extensive surveys carried out by Selikoff and his colleagues (Selikoff et al 1965, Selikoff 1965) showed that many insulators had developed changes associated with asbestosis.

The increasing numbers of insulators accepted by the Pneumoconiosis Medical Panel as having asbestosis, and the papers of Elmes (1966), Anton (1967) and Leathart (1968) showed that British insulators were also showing the effects of exposure to asbestos. The radiographic survey carried out by Sheers and Templeton (1968) in Devonport Dockyard confirmed that pulmonary fibrosis was

occurring in the men heavily exposed to asbestos while large numbers of men not thought previously to have been at risk showed signs of pleural abnormality associated with asbestos exposure.

The increasing numbers of mesothelial tumours associated with asbestos exposure, and reports of increased incidence of bronchial and other cancers occurring in insulation workers (Mancuso and Coulter 1963, Selikoff, Churg and Hammond 1964, Elmes 1968) helped to focus attention on the problems associated with asbestos.

The present survey has shown that, in the Dockyard, men were often exposed by working with, or near, other men who were handling asbestos products. The men directly involved with asbestos materials were given some form of respiratory protection and came under a scheme for medical supervision; those working nearby were unprotected.

The results of the clinical survey (Chapter 4, pages 140-278) show that the protection afforded to the laggers and sprayers was insufficient to prevent sizeable proportions of them from developing asbestosis. Consideration of the dust concentrations to which they were exposed (Chapter 3 , pages 84-139) partly explains why so many were affected.

The respirators provided by the Dockyard to protect these men varied in efficiency. Even when they were of good fit they would only have been between 95-98% efficient, and until recently there was no attempt to see that a man obtained a respirator to suit his face. It must be admitted that respirators are uncomfortable to wear, and attempts to make them more comfortable, such as the use of stockinet around the face piece, again decreases the efficiency of the respirator. If the respirator is not worn

then its efficiency is nil, and the reluctance to wear respirators, and inadequate encouragement of men to wear them, must also play a large part in the causation of asbestosis in these men.

Respirators were usually worn if the dust clouds were intense and prolonged, as in spraying or stripping asbestos, but were not worn for short periods of exposure even though the dust concentrations would have been high. This is understandable because a man would remove a small amount of asbestos from a pipe or steel plate without using respiratory protection instead of first walking ashore to obtain a respirator.

Similarly, men removing sprayed crocidolite asbestos would take their respirators off periodically to talk, or smoke, in the dust laden compartment, even though they were forbidden to eat in the compartment which they were stripping. Meals were usually taken in adjacent compartments, and the dust sampling survey has shown that the dust concentrations in these adjoining compartments were very high.

The protection afforded to the men working with asbestos was inadequate, but there was no protection for the men working near them. Most of these men in other trades would try to avoid the compartments with intense dust clouds, but they were often obliged to work in them. In many cases these men might have been unaware of the risk involved in working in a passageway outside a compartment which was being stripped of asbestos. Without a special lighting technique this dust is not easily seen.

It might be argued that the medical supervision of the men exposed to asbestos was not of much value, but the detection of a dozen men thought to have clinical evidence of asbestosis at their annual medical examination in 1965 was the start of this comprehensive study (see page 7). The use of

70 mm chest films with a technique designed to concentrate attention on the lung apices was unsatisfactory, and a return to full size radiographs for the examination of asbestos workers was made at the outset of this study.

It has been explained that the dangers associated with the use of asbestos had been considered by Naval Medical Officers working in the Dockyard Occupational Health Service, but because few men were found to be affected by the dust little or no action was taken to improve working conditions.

The alteration of work schedules, provision of costly protective equipment, and changes in material specification cannot be undertaken lightly. It is argued that all these precautions cost money and slow the rate of production. Thus, to achieve such changes and preventive measures, evidence must be produced to show that they are necessary. Such evidence was available early in the present survey, and, when it was presented to them, the Medical Department and the Management responsible for HM Dockyards (Ministry of Defence, Navy) acted with commendable speed in improving work methods, protective equipment and medical supervision.

There are two main reasons why there was insufficient evidence of the harmful effects of asbestos in Naval Dockyard workers before 1965. The first, and most important reason lies in the nature of the diseases associated with asbestos, and the length of time the men have been exposed to relatively high dust concentrations. It must be remembered that it was not until about 1944 that very large amounts of asbestos were used in Naval Dockyards. From then, until the early 1960's, enormous amounts of crocidolite and amosite asbestos were sprayed on or stripped off deckheads, or put on pipes and machinery. Extensive refits of large warships resulted in the widespread exposure of large

numbers of men to intermittently high dust concentrations. Thus it was only after 20 years or so that most of the effects of this asbestos exposure started to become apparent. This long latent period is shown in several previous studies. Selikoff (1965) showed that the proportions of insulators affected by the disease increased with time, and there were relatively few men affected with under 20 years exposure. Similarly, Newhouse (1969) has shown that the incidence of malignant disease associated with asbestos increases after 16 years of severe exposure to the dust.

In 1965 sufficient time had elapsed for Dockyard men to show signs of the disease, and this leads to the second reason why they were discovered to have the disease, namely more intensive medical investigation and improved methods of examination.

While it is true that the initial twelve men were discovered to show signs of asbestosis by the use of the stethescope, the diagnosis was only confirmed after careful chest radiography and lung function tests had been carried out. Examination of men known to have been asbestos sprayers revealed more cases, and these findings resulted in the organisation of the present survey and that of Sheers and Templeton (1968).

Dust Sampling Survey

The description, in the present survey, of the processes involving asbestos and the men exposed to it, is sufficient to explain why so many men have been affected, but the dust sampling survey confirms the degree of risk to which they have been exposed. The dust concentrations during the spraying of asbestos were exceedingly high, and this must be considered as a most hazardous operation. The process is no longer in use in Naval Dockyards,

but high fibre concentrations still occur during the stripping of sprayed asbestos material. Most of this material was of crocidolite asbestos which is considered to be the most dangerous variety. The results of the dust sampling survey showed that high fibre concentrations existed in compartments and passageways often remote from that in which the stripping was taking place. This was so even with the extensive precautions taken in an attempt to control the spread of the dust in 1967. Before that date there was no attempt to contain the dust, and asbestos debris littered the decks for long periods of a refit, thus exposing very many men to the dust.

Dust sampling showed that it was possible to remove sprayed asbestos and to clean the compartments so that no asbestos was detectable in the atmosphere of the cleaned compartment. This meant that other work could then proceed without danger to men who would not then need to wear protective equipment.

Machinery spaces, engine rooms, and boiler rooms could similarly be stripped of their insulation, and while this caused very high asbestos dust concentrations whilst it was being done, careful cleaning ensured that the compartments were afterwards safe to work in without respiratory protection.

The application of insulating materials was shown to cause low general atmosphere fibre concentrations, but concentrations of 50 fibres per c.c. or more occurred locally at the point of application. This emphasized the need for men applying the insulating materials to continue to wear their respirators even when using materials with low asbestos content.

Other miscellaneous processes were shown to be associated with local fibre concentrations of over 50 fibres per c.c. These included mixing asbestos cement in a bucket, fitting asbestos rope, sweeping up asbestos fibre in the

mattress shop and brushing slag from asbestos cloth used by welders. Joiners were exposed to high concentrations when removing friable acoustic board, and men unpacking insulating sections were also exposed to concentrations over 50 fibres per c.c.

Some processes were found to give dust concentrations of less than 1 fibre per c.c. suggesting that it was not necessary for men to wear respiratory protection. The manipulation of dust suppressed asbestos cloth was shown to give rise to dust concentrations of less than 1 fibre per c.c. - except when it was brushed or chipped. This suggested that welders and burners could safely use it without wearing respirators provided that they did not chip or brush the cloth.

It is of interest to record some results of samples taken inside storehouses and workshops which were sprayed with amosite asbestos. These sheds were roofed with corrugated iron and sprayed, inside, with amosite asbestos to a thickness of 1-2 inches. General atmosphere counts were between 1.5-2.0 fibres per c.c. Whenever the surface of the sheds were disturbed by being brushed against by the men's clothes, or by stacking sheets of timber against the walls then local dust concentrations of between 2.0-7.5 fibres per c.c. were recorded. Birds had nested in the material, and asbestos fibre was scattered over shelves and stores. When the stored packages were moved local concentrations of between 4.0-7.5 fibres per c.c. were created.

A survey of dust concentrations in buildings incorporating asbestos materials in their construction reported by Byrom et al (1969) showed fibre concentrations of between 0.00 and 0.08 fibres per c.c. That report concluded that buildings incorporating asbestos products in their construction

were not likely to constitute a hazard to health. The report did not state whether or not the asbestos materials were exposed to the atmosphere.

The few results from the present survey show that when sprayed asbestos is exposed to the working environment there is always the risk of asbestos fibre being liberated from its surface. This occurs with vibration, physical contact and unlikely events such as the nesting of birds. The solution of this problem was to coat the asbestos fibre with hard setting cement and to paint the cement surface.

Preventive Methods and Dust Sampling Results

Consideration of the dust sampling results helped in the appreciation of the degrees of risk associated with different work processes, and in the formulation of methods to reduce these risks. For example, stricter control was exercised over the isolation of work involving the stripping of blue asbestos. Greater care was taken in cleaning out compartments stripped of asbestos, and steel plates were spray painted to stick down any remaining loose fibre. Air fed hoods and impervious overalls were provided for all men who had to enter that part of the ship in which blue asbestos was being removed, and special changing, washing and eating places were provided on the ships for these men. Because dust sampling showed that showers did not wash off all traces of asbestos from the impervious overalls a man was provided to clean these thoroughly before they were used again. He was supplied with an impervious suit and an air fed respirator.

Similar precautions are now taken when any large amount of work occurs involving asbestos materials. Any process where the general atmosphere fibre count is likely to be 50 fibres per c.c. or more is regarded as requiring the full precautions to protect individuals from exposure to the dust.

Lesser dust concentrations, which are often found during small jobs lasting for short periods of time, or during the application of pipe lagging, do not require such extensive protective measures, and bri-nylon overalls together with dust respirators, or sometimes positive pressure power respirators, are sufficient.

Dramatic reductions in general atmosphere fibre concentrations have resulted from the policies introduced after consideration of the dust sampling results. Thus for the six months May-October 1969 (inclusive), for engine rooms there were 343 samples with less than 1 fibre per c.c. and only 17 samples with more than 1 fibre per c.c. The mean of the 17 samples was 2.9 fibres per c.c. For boiler rooms, over the same period of time, there were 273 samples with less than 1 fibre per c.c. and nine (mean 2.7 f/c.c.) with more than 1 fibre per c.c. In smaller compartments there were 327 samples with less than 1 fibre per c.c. and 25 samples (mean value 31.8 f/c.c.) with more than 1 fibre per c.c.

These figures show that, for the application of pipe lagging, the dust concentrations are capable of being reduced to a level which is approaching that suggested by the British Occupational Hygiene Society Sub-Committee on asbestos (1968), as being unlikely to cause asbestosis. It must be remembered that the Committee specifically referred to chrysotile asbestos, and it is not known how much amosite asbestos was present in the Dockyard samples. It is

possible that over 50% of the fibre in the Dockyard samples would be amosite asbestos. The results show that considerable care must continue to be exercised in making workpeople involved in these processes wear the protective clothing and respirators provided for them. The figures also show that there are higher concentrations to be found where men are working in small, confined compartments.

The reduced dust concentrations are the result of better working methods and the introduction of more material containing little or no asbestos. Men are more careful in handling insulating materials, and debris is regularly and thoroughly removed from the compartments. The addition of a dust suppressant to asbestos cloth by the manufacturers has been successful in reducing the amount of airborne asbestos fibre.

The use of some of the alternative materials has presented problems. Some glass fibre cloths and rope have caused such intense irritation of the skin and mucous membranes that men refused to work with them. This has stimulated urgent research and development by the manufacturers to produce a product which is technically satisfactory, and acceptable to the workman. There are now several different varieties of glass fibre cloth which do not cause skin irritation when handled.

Most of the other problems associated with alternative materials have been technical. The material has either been too brittle, or did not have the same thermal insulation efficiency as asbestos, or was altered by prolonged heat and vibration leading to impairment of its insulating properties.

Another problem is that several of the so called "asbestos free" insulating materials have been found to contain appreciable amounts of fibrous

material which is optically indistinguishable from asbestos. Dust sampling technicians have been instructed to report these fibres as asbestos unless they are certain that they are looking at glass, vegetable, or man made fibres. It is not yet known what alternative fibres have been introduced into some of these insulating materials. If the fibres are similar to asbestos then it is not unlikely that they may possibly have similar harmful characteristics. In any event the new materials produce intense particulate dust clouds, mainly magnesia or calcium silicate, so that respiratory protection will continue to be essential for men applying or removing them.

Clinical, Radiological and Physiological Survey

The evidence obtained from this survey is such that it is difficult to fully explore the complex interactions of the many variable factors which affect the individuals forming the study population. I have presented the data in a manner which attempts to examine some of these inter-relationships. More detailed and complex analyses are being undertaken by Mr. P.D. Oldham, and will be reported at a later date. The survey forms a base line for future studies, and those studies will be designed to examine the progression of the diseases associated with asbestos in this population.

The results show that there are definite relationships between the clinical, radiological, and physiological changes associated with asbestosis and the intensity and duration of exposure to asbestos. It is clear that the preventive measures enforced before 1966 were insufficient to prevent many laggers and sprayers developing changes associated with asbestosis. It is also obvious that men intermittently exposed, and those more frequently exposed to low dust concentrations, have developed changes associated with asbestos exposure.

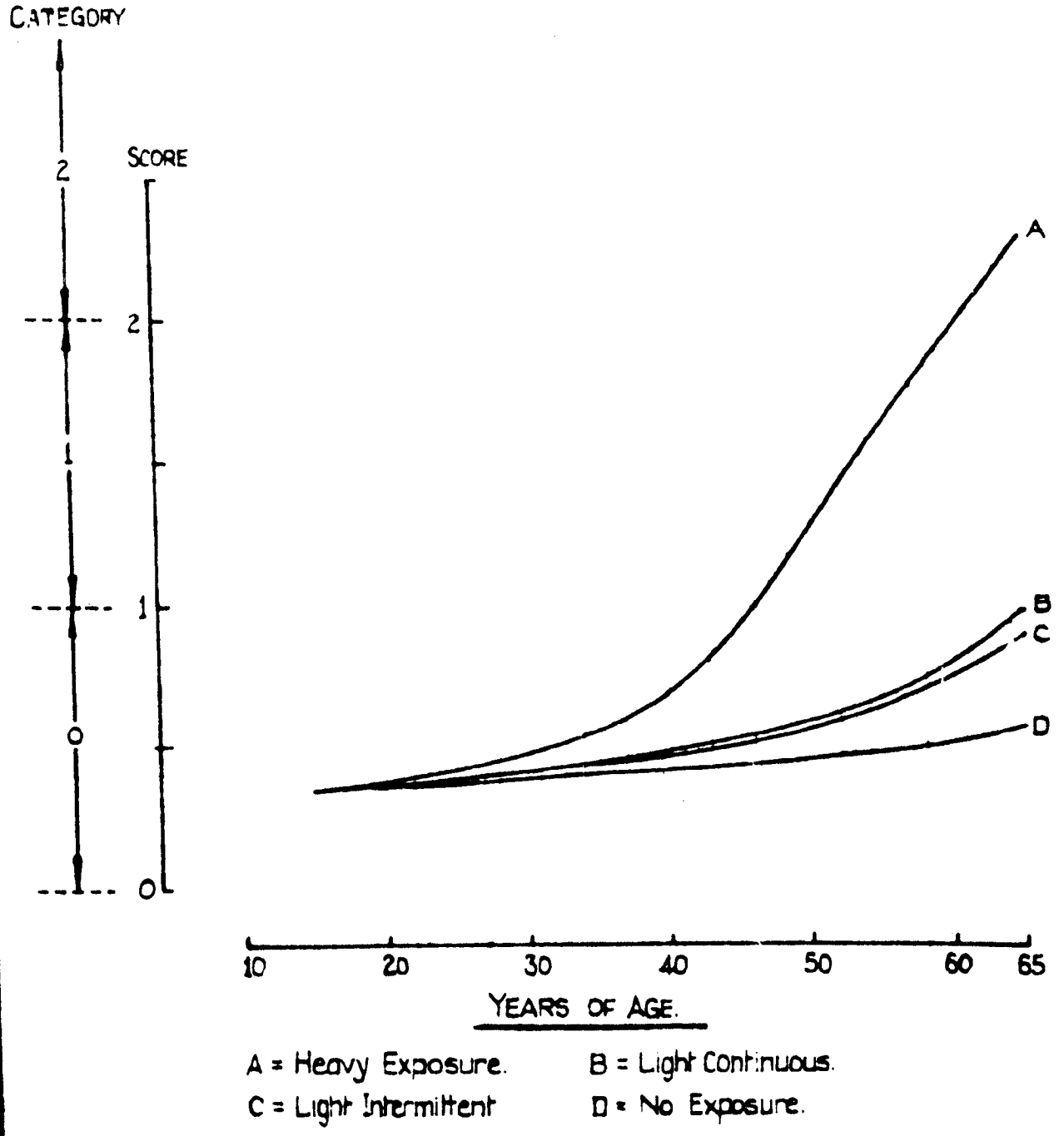
Productive morning cough, dyspnoea, basal rales and finger clubbing are the symptoms and signs which are associated with the intensity and duration of exposure to asbestos in this population. The basal rales are quite characteristic and, when heard, are important evidence suggesting pulmonary fibrosis. There is sometimes difficulty in differentiating between the rales and dry pleural friction creaks. When I am in doubt it is my custom to regard these noises as being of pleural origin. In the established disease the rales extend over most of the lung fields, and may often be the only evidence of any underlying pathology. The data from this study suggests (figure 4.17, page 186) that when these combinations of symptoms and signs occur in men with normal radiographs, then they are accompanied by the lung function changes associated with asbestosis.

The radiographic abnormalities occurring in the survey population have been shown to be related to the intensity and duration of exposure (figure 4.13, page 178). Another analysis by Mr. P.D. Oldham shows (figure 6.1, page 336) that although men with light continuous, and intermittent exposure, have radiological appearances which are hardly distinguishable from each other, they are markedly different from those men with no exposure at all. The men with heavy exposure show the widest deviation from the men without exposure.

The radiographic changes seen in this population are often ill defined deviations from normality. Irregular small opacities, more noticeable over the lower zones are the most typical early changes. The men with well established disease have extensive irregular opacities, with many linear shadows, sometimes forming a reticulation over both mid and lower zones. In these moderately advanced cases the outlines of the diaphragm and pericardium are indistinct and irregular, and there is usually other evidence of pleural fibrosis or calcification.

FIG 6.1

REGRESSION OF IRREGULAR SMALL OPACITY SCORE ON AGE, FOR A MAN OF HEIGHT 1·7m., WEIGHT 74 kg, EXPOSURE BEGINNING AT AGE 15 YEARS. READINGS OF CATEGORY 0/0 BY ALL OBSERVERS TAKEN AS "LESS THAN THE SCORE FOR ONE 0/1, THREE 0/0."



A = Heavy Exposure. B = Light Continuous.
C = Light Intermittent D = No Exposure.

The most common pleural abnormalities in this population are small areas of hyaline pleural thickening seen on the lateral chest walls. Pleural calcification is relatively uncommon, being seen in only eleven men. Extensive pleural fibrosis, with or without effusion, is only seen in three men. The varieties of pleural abnormalities seen in Devonport Dockyard workers have been described by Mackenzie (Mackenzie and Harries 1969), and he has developed a radiographic technique which is of great value in the examination of the pleura. This technique is an improvement on the standard method of obtaining oblique views of the chest, and shows the pleura clearly in those cases where there is doubt on the posterior-anterior film as to whether or not there is pleural abnormality.

The U.I.C.C. Cincinnatti classification is useful in its attempt to provide an objective assessment of radiographic abnormality. It is not easy to use until the observer has become accustomed to objective recording of the shadows he sees, rather than the more subjective interpretation of these shadows. It would appear, from the data presented in this survey, that this classification will form an important method of recording the radiographic appearances of men exposed to asbestos. The classification should help in the prospective surveys being planned to study the progression of the disease, and should also help to make more valid comparisons between studies of different populations, if it is generally accepted and used.

The physiological tests used in this survey have been shown to be related to the intensity and duration of exposure to asbestos, and also to the degree of radiographic abnormality. There is some evidence (figure 4.20, page 190) that estimations of the ventilatory cost of exercise, and of the transfer factor may also be correlated with the degree of dyspnoea present,

and this might be an important factor in assessing the disability of the men.

It is difficult to estimate the degree of disability in many of the cases showing evidence of abnormalities due to asbestos. Disability pensions are awarded where there is evidence suggesting pulmonary fibrosis, and the amount of the award depends on the estimated disability. Men with evidence of pleural abnormality without obvious pulmonary fibrosis are not usually awarded pensions, but in some cases these men might be considerably disabled. More work is required to see whether or not the estimation of the ventilatory cost of exercise will be of real value in assessing disability. In my limited experience this test does seem to give useful information about the degree of effort dyspnoea experienced by the patient.

These tests should not be considered in isolation when examining men suspected of having asbestosis. The results from the tests of ventilatory capacity usually show a restrictive defect with no evidence of airways obstruction; the transfer factor is usually reduced; and the exercise or standardized ventilation is increased in those men who are developing pulmonary fibrosis. Men with diffuse pleural fibrosis show a similar pattern, but with less of a reduction in the transfer factor. Pleural calcification is seldom accompanied by any marked changes in lung function, excepting those cases of extensive constrictive pleural calcification, in whom there is usually a restrictive defect, but little change in transfer factor. It should be remembered that while small differences seen in the grouped data can be significant, (figure 4.8, page 170) small differences between individual cases are usually not significant.

The general situation with regard to the diseases associated with exposure to asbestos in Devonport Dockyard up to July 1969

Asbestosis

Eighty-five Dockyard employees have been awarded disability pensions for asbestosis by the Pneumoconiosis Medical Panel. This number includes the 36 men described earlier in this report (Table 4.XXXVIII, page 239). The numbers of men in occupational groups are shown in Table 6.I (page 350). The laggers have the largest number of affected men, followed by the sprayers, but 42 of the 85 men come into the category of "intermittently exposed". The present survey has shown the likely dust concentrations these men have been exposed to over the last 25 years.

Few cases were known before 1965 (Table 6.II), and the large increase in the number of cases since then reflects both the time interval between exposure and development of the disease, and the increasing awareness of the problem which has been stimulated by the present research work. New cases are now presenting at this Unit at the rate of about one every month.

Pleural Abnormalities due to Asbestos Exposure

These abnormalities include cases with calcified pleural plaques, limited hyaline pleural plaques, and extensive pleural fibrosis. Pleural calcification is relatively uncommon, and occurs in the older men with long exposure to relatively low dust concentrations. Hyaline pleural plaques are the commonest manifestation and appear usually after 20 years from first exposure to the dust.

There are increasing numbers of younger men (35-45 years) presenting with unilateral or bilateral pleural reactions which are often accompanied

by pleural effusions. The effusions usually resolve leaving an area of pleural fibrosis. In some cases the pleural abnormality is progressive. Extensive investigations have not demonstrated any infective or traumatic explanation for these reactions. The one thing the men have in common is that they have been exposed intermittently to asbestos since the age of 15 or 20 years. Some of these men have a restrictive ventilatory defect with a reduction in transfer factor, and the men with extensive pleural reactions are disabled.

A central register of cases of pleural abnormalities is being prepared in Plymouth, and this register will combine the data now available at the Plymouth Chest Clinic, this Research Unit, and other sources, so that a detailed prospective study can be undertaken. There are at present more than 450 men, in Plymouth, known to have pleural abnormalities. Research is urgently needed in order to determine the true significance of these conditions and to find out which of them may be associated with progressive disabling disease, or with malignancy. It is to be hoped that the great majority of these cases will not suffer disability or develop malignant disease of the lung or pleura.

Table 6.III compares the symptoms and signs occurring in 156 men with pleural abnormalities with those recorded in 81 men with "certified" asbestosis. The comparison of lung function values is given in Table 6.IV. These findings are similar to those previously reported in this survey (see Clinical section, pages 140-278). The two groups are obviously highly selected in that the cases of asbestosis were diagnosed by the Pneumoconiosis Medical Panel, and the men with pleural abnormalities were those for whom I had adequate lung function data in July 1969. Because of their selection it is not profitable to draw many conclusions from this comparison. A

prospective physiological study of all men known to have pleural abnormalities has been designed to study these manifestations.

Pleural Mesothelioma

A histological diagnosis of pleural mesothelioma has been confirmed in 29 Dockyard employees. Most of these men have presented at the Plymouth Chest Clinic, and I am grateful to Dr. G. Sheers and his colleagues for supplying data on their patients. I have only included those cases in which the diagnosis has been confirmed by Dr. J.C. Wagner, Pneumoconiosis Research Unit, Penarth.

It can be seen, from Table 6.V, that none of the affected men is a recognised asbestos worker. The lagger's mate would now be a registered asbestos worker and properly protected from the dust. The other men should now not be exposed to asbestos, if the regulations are properly enforced.

Most of the men have been exposed to asbestos for more than 25 years. One man had only two years exposure to the dust, but it occurred 30 years previously. The time from first exposure to asbestos was more than 30 years for 15 of the 19 cases for whom this information is available.

There is only one known case of pleural mesothelioma in Plymouth in a person not employed in the Dockyard. This man was employed as a railway engine fitter, and frequently had to remove or replace asbestos insulating material.

The present data suggest that the number of cases of pleural mesothelioma is increasing, and this is probably true. It is only since 1965 that so much interest has been taken in this condition, and it is possible that some cases might previously have been diagnosed as bronchial carcinoma.

No case of peritoneal mesothelioma in Devonport Dockyard employees has yet been confirmed.

Lung and Gastro-intestinal Cancer

The proportional mortality study has several disadvantages, but the data suggest that, up to 1968, there is no evidence of an increased incidence of lung or gastro-intestinal cancer deaths in men employed in Devonport Dockyard. It is likely that the period of time that the men have been exposed to asbestos is not yet long enough for any increased incidence to become apparent.

In her study of asbestos factory workers Newhouse (1969) has shown that the increase in lung cancer incidence did not become obvious until the workers had been heavily exposed for more than 16 years. The men, in her study, who might have had similar exposure to the majority of intermittently exposed Dockyard workers were classified as having moderate exposure. The low-moderate group of asbestos factory workers did not show any increased incidence of lung cancer even up to 31 years after first exposure to asbestos.

Data for Dockyard laggers are not complete, so that it is not yet possible to compare the mortality of these severely exposed men with those reported by Newhouse. The cause of death of 12 of the very heavily exposed asbestos sprayers is known, and 6 of them died of malignant neoplasms.

More work is required on this problem, but it seems possible that we may observe an increasing incidence of malignant disease occurring first in the most heavily exposed, and possibly, later in the less heavily exposed Dockyard population. This is speculation, but the proportional mortality study forms a satisfactory base line for a long term prospective study.

The social effects of disease associated with asbestos on Dockyard workers.

The data which have been presented in this study are disturbing enough in themselves, but they do not give much idea of how these diseases affect the lives and work of the men.

The pulmonary fibrosis associated with exposure to asbestos is a progressive disease, and produces increasingly disabling effort dyspnoea. The men find it more and more difficult to perform their work, especially if this involves heavy physical effort, climbing up and down dry docks, or negotiating the many vertical ladders aboard ships. They find it difficult to get to work in the winter. Many rise at 5.30 a.m. in order that the distressing early morning coughing bout is over by the time they report for work at 7.30 a.m. Cold, damp mornings increase their discomfort, and intercurrent chest infections make their disablement worse.

The Dockyard Medical Service consults with management in an attempt to find such men suitable work. Such work would be away from dusts and fumes, not requiring heavy physical effort, and preferably ashore in a workshop. These jobs are becoming increasingly difficult to find because of the large numbers of men partially disabled for other reasons.

Changing the job usually, but not always, results in a reduction in wages, and if this happens early in life the total financial loss can be considerable. It is true that the men receive a disablement pension if they have been accepted by the Pneumoconiosis Medical Panel as suffering from asbestosis, but this, even with the special Hardship allowance, does not often make up for the reduction in pay. The Ministry of Defence recognises this and takes it into account when considering any claim for compensation

which might be brought by men suffering from these diseases.

It is usual not to inform men that they are suffering from pleural mesothelioma, but to explain to them that they have asbestosis or a pleurisy due to asbestos exposure. The relatives are fully informed of the situation, so that they may be forewarned of the poor prognosis. Patients are given assistance in seeking examination by the Pneumoconiosis Medical Panel so that they may receive a disability pension.

Men with pleural fibrosis or pleural calcification are advised to regard themselves as being normal in every way, but are encouraged to give up smoking. If their work involves exposure to asbestos then they are offered alternative employment. These men are not considered to be suffering from "asbestosis" unless they show signs of disablement. Those men who are disabled because of pleural abnormalities are helped to seek examination by the Pneumoconiosis Medical Panel.

Conclusions

Increasing numbers of men in Devonport are developing diseases associated with exposure to asbestos. Another survey (Harries et al 1969) suggests that this is likely to happen in other Naval Dockyards.

Consideration of the working methods and the results of the dust sampling survey has indicated the degrees of risk involved. This has resulted in the formulation of a new Code of Practice for all work with asbestos materials in Naval Dockyards. Progress has been made in the provision of better protective equipment and changing facilities for the men. Increasing research is being undertaken to find suitable alternative materials to replace asbestos. Dust monitoring teams have been set up in each Naval Dockyard so that a check can be made on the efficacy of the protection provided for the workmen in different work situations.

The clinical study has shown that the disease processes are related to the intensity and duration of exposure. The clinical, radiological, and physiological abnormalities occur more frequently in those men most heavily exposed to asbestos. It is, therefore, reasonable to suggest that the reductions in dust concentrations that have been achieved, together with the better protective clothing that has been provided, should eventually greatly reduce, if not eliminate, pulmonary fibrosis in laggers and other registered asbestos workers. The men previously intermittently exposed to asbestos will, in future, have virtually no exposure to asbestos as long as the new regulations laid down by the Ministry of Defence (Navy) are properly enforced.

It is a regrettable fact that these preventive measures will probably have little beneficial effect on those men who have previously been exposed to the dust over the last 25 years. This is one reason why more medical research is required. The survey has attempted to evaluate the problems and to suggest avenues for future research.

The results of the survey show that the new radiographic classification proposed at Cincinnatti in 1968 is of use in the study of men exposed to asbestos. The usefulness of the method can only be fully exploited by means of rather complex calculation of scores for irregular opacities. A method of calculating a score for pleural abnormalities has not yet been devised. Evaluation of both pleural and parenchymal abnormalities would be of great value, and further work is proceeding to achieve this.

While the new classification seems to be of value in epidemiological studies, the interpretation of individual radiographs remains a difficult problem. Such interpretation is probably of the greatest value when the radiograph is considered together with the history of the patient, his

exposure history, physical signs, and lung function results. The difficulties of the interpretation of radiographs of men with pneumoconiosis, the variation due to differing techniques, and the importance of not relying solely on the x-ray to make a diagnosis have been explained by McLaughlin (1962). He suggested that the occupational history might prove to be the most valuable indicator of the possibility of pneumoconiosis. It should be remembered that Ramazzini (1713) suggested that it was important to find out the patient's occupation before attempting to make a diagnosis.

The data presented in this survey confirm the observations of others that disease associated with asbestos is occurring in men in whom exposure to asbestos was previously unsuspected. A careful occupational history is thus of great importance in attempting to assess the likely exposure to asbestos. Men working at most unlikely trades have been shown to have been exposed to the dust in the Dockyard. It is reasonable to suggest that, until the new Code of Practice for HM Dockyards came into force in April 1969, any employee who had worked in ships under refitting conditions many have been intermittently exposed to high concentrations of asbestos.

Clinical examination remains a very important part of the investigation of men exposed to asbestos. The survey has shown that the development of basal rales may precede radiographic abnormalities. This has previously been suggested by Leathart (1968c).

Lung function tests are now an equally important part of the assessment of men working with asbestos. The measurement of ventilatory capacity (FVC and $FEV_{1.0}$), lung volumes (RV and TLC) and the transfer factor (T_L and T_L1) are essential for the examination of men suspected of having asbestosis. The ventilatory cost of exercise (SV and EV) may prove to be of value in estimating the disability caused by the disease.

It is hoped that more useful information correlating the clinical, radiological, and physiological changes in this population with their exposure to asbestos will be obtained from further analysis of the data.

The problems facing those responsible for the health of Dockyard employees exposed to asbestos fall into two groups. Firstly, those concerned with the medical supervision of men working with insulating materials which will contain less and less asbestos. Secondly, those associated with the detection, and possible treatment, of diseases due to asbestos previously inhaled by the men working in the Dockyard.

It is not possible to consider these problems in isolation and the proposals for future work in Naval Dockyards includes both the better medical supervision of registered asbestos workers, and an intensive research project into most of the aspects of the diseases due to asbestos.

Registered asbestos workers are to continue to have an annual medical examination. This will consist of a clinical examination, full size chest radiograph and estimation of the Forced Vital Capacity (FVC) and Forced Expiratory Volume in 1 second ($FEV_{1.0}$). At present it is not considered practicable to set up in each Dockyard the apparatus required to estimate lung volumes and the transfer factor.

It is hoped that within three years it may be possible to make available to each Naval Dockyard a mobile physiological unit which would be able to carry out these, and other measurements.

Chest radiography will be made available to every Dockyard employee, and this is both part of the general improvement in medical supervision, and part of the research programme.

An extensive research programme is proposed and the proposals are based on the experience gained from the present survey and those of Sheers and Templeton (1968) and Harries et al (1969). The aims of the work are to study the natural history of the diseases associated with asbestos, with particular reference to factors affecting predisposition, progression, and prognosis. It is hoped to evaluate the safety of the existing environmental conditions of the Dockyards following the introduction of the New Code of Practice for work involving asbestos, by determining the attack rate over future years.

It is proposed that a radiographic survey of the total Dockyard population will be undertaken every two years. This is an extension of the use of the Royal Naval Medical Service Mass Mobile X-ray Units which have been improved and increased in number for this purpose. It is hoped that prospective studies of the men found to have early parenchymal or pleural abnormalities will help to clarify the natural history of these diseases.

A long term mortality study is planned, which will examine the causes of death of the 40,000 employees in the four Naval Dockyards in the United Kingdom over the next 10-15 years.

The work will be co-ordinated by the Medical Research Unit at Devonport (which is part of the Institute of Naval Medicine) in conjunction with the Medical Research Council Pneumoconiosis Research Unit.

In Devonport more extensive physiological studies will be undertaken, and the re-examination of the population described in the present report is now almost complete. It is hoped that this, and subsequent examinations of this population will provide useful information about the progression of the diseases, and of the most appropriate methods of examining these subjects.

The immunological status of this population is now being determined by Dr. M. Turner-Warwick of the Brompton Hospital.

The most important part of the research programme is probably the study of men with pleural abnormalities. It has been shown that there are large numbers of such men, and that the abnormalities are occurring in relatively young men. A prospective study of all the men showing these changes in each Dockyard is planned. This will involve the close co-operation of many different disciplines, and will be designed to attempt to discover which changes are associated with the development of disabling fibrosis, or with malignant changes, and to explore the possible means of treatment.

I consider that it is reasonable to conclude this report on a note of cautious optimism. The problems have been stated, solutions designed to prevent future disease have been suggested and are being implemented. It has been recognised that this will not sufficiently help those who have already been exposed to the dust, and research into possible ways of treating the diseases due to asbestos has been proposed. It is to be hoped that the efforts to prevent the diseases, and the search for methods of treatment of the diseases due to asbestos may be rewarded with success in the not too distant future.

TABLE 6.I

Occupations of 85 Men with Asbestosis

Occupation	Number of Men
Laggers	26
Sailmaker Laggers	2
Asbestos Sprayers	15
Skilled Labourers Afloat	8
Electrical Fitters	7
Engine Fitters	4
Caulker/Rivetters	4
Welders	3
Shipwrights	3
Small Ship Engineers	2
Boilermakers	2
Painters	2
Joiners	2
Coppersmith	1
Telephone Operator (previously Lagger)	1
Plumber	1
Slinger	1
Shot Blaster	1
TOTAL	85

TABLE 6.II

Annual Number of cases of asbestosis in Devonport Dockyard accepted by the Pneumoconiosis Panel

Year	Number of Cases Accepted
Before 1963	3
1964	1
1965	4
1966	10
1967	46
1968	14
1969 to 30th June	7
TOTAL	85

TABLE 6.III

Comparison of symptoms and signs in 156 men with pleural changes and 81 men with asbestosis.

Symptom	Pleural Changes		Asbestosis	
	No. of Men	%	No. of Men	%
Cough	65	42%	56	69%
Sputum	66	42%	52	64%
Dyspnoea Grade 2	76	49%	39	48%
Grade 3 or more	12	8%	38	47%
Past Illnesses				
Bronchitis	50	32%	31	38%
Pneumonia	23	15%	10	12%
Pleurisy	30	19%	14	17%
No. of Smokers	108	69%	55	68%
Signs				
Rales	50	32%	74	91%
Clubbing	15	10%	37	46%

TABLE 6.IV

Comparison of Lung Function Results for Men with Pleural Thickening and those with Asbestosis

	(X) No. Examined Pleural Changes	(Y) No. Examined Asbestosis	(X) Mean	(Y) Mean	(X) s.d.	(Y) s.d.
Age	156	81	53.70	56.90	9.05	8.50
Height	156	81	168.40	169.20	7.09	6.63
Years of Exposure	156	81	23.50	23.60	10.14	8.89
Years since 1st Exposure	156	81	27.60	27.20	10.11	9.36
FVC	156	81	3.42	2.91	0.66	0.70
$FEV_{1.0}$	156	81	2.52	2.09	0.62	0.59
$FEV_{1.0}/FVC\%$	156	81	73.40	71.80	9.22	10.60
TLC	156	75	5.56	5.00	0.93	0.96
IC	156	75	2.35	1.87	0.60	0.53
RV	156	75	1.82	1.83	0.58	0.60
RV^1	156	75	1.56	1.37	0.40	0.41
RV^1/RV	156	75	0.89	0.78	0.22	0.21
VC	156	75	3.73	3.16	0.70	0.69
T_L	156	73	25.24	18.96	5.41	5.18
$T_L{}^1$	156	80	24.10	16.99	5.56	4.78
RV/TLC	156	75	32.70	36.50	7.74	8.43

TABLE 6.V

Details of 29 Cases of Pleural Mesothelioma

Occupation	Date of Death	Age	Years since First Exposure	Years of Exposure
1 Boilermaker's Mate	1960	66	30+	30+
2 Lagger's Mate	1964	58	30	30
3 Crane Driver	1964	50	34	34
4 Boilermaker	1965	64	Not Known	At least 14
5 Shipwright	1965	56	29	29
6 Shipwright	1965	64	40	34
7 Welder	1965	51	Not Known	
8 Slinger	1966	70	35+	22+
9 Boilermaker's Mate	1966	48	31	24
10 Engine Fitter	1966	69	44	44
11 Shipwright	1966	66	Not Known	
12 Shipwright	1967	64	46	33
13 Engine Fitter	1967	62	30	2
14 Welder	1967	39	20	20
15 Welder	1967	59	35	35
16 Boilermaker	1967	64	46	46
17 Welder	1968	63	42	42
18 Electrical Fitter	1968	61	29	29
19 Caulker/Riveter	1968	49	30	30
20 Shipwright	1968	66	50	45
21 Shipwright	1968	80	Not Known	
22 Machinist	1969	79	Not Known	
23 Electrical Fitter	1969	56	30	18
24 Engine Fitter	1969	76	25+	25
25 Boilermaker	1969	88	Not Known	
26 Engine Fitter	1969	79	Not Known	
27 Welder	*	61		
28 Electrical Fitter	*	61		
29 Shipwright	*	45		

*Biopsy Diagnosis

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SUBSIDIARY PAPER

SECOND INTERNATIONAL CONFERENCE ON THE
BIOLOGICAL EFFECTS OF ASBESTOS
DRESDEN 22ND TO 25TH APRIL 1968

Summary of paper

The Prevention of Asbestosis in Naval Dockyards

by P.G. Harries

This paper presents a short account of the measures taken in the Naval Dockyards of the United Kingdom to minimise the hazards associated with the many processes involving asbestos containing materials in ship-building and ship-repairing.

These follow the accepted principles of industrial preventive medicine, namely, substitution, segregation, dust suppression, protective clothing with various forms of dust respirators, and medical supervision of employees.

It has been possible to find substitutes for a variety of asbestos containing materials and, by suitable planning, it has been found possible to segregate most of the processes causing high concentration of asbestos dust.

Improved dust respirators, airline respirators, and positive pressure power respirators have been found acceptable to the employee working in high dust concentration.

A brief description is given of the medical supervision of the workers and of the medical research projects now being carried out in one Naval Dockyard.

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Devonport.

18th December 1967.

SECOND INTERNATIONAL CONFERENCE ON THE BIOLOGICAL EFFECTS OF ASBESTOS

The Prevention of Asbestosis in Naval Dockyards

P.G. Harries, Medical Research Council,

Devonport Dockyard

Many different types of asbestos containing materials are still used in shipbuilding for heat, sound and electrical insulation, and many authors have drawn attention to the exposure of workers to asbestos in this industry (1, 2, 3, 4, 5, 6). Dust is produced in many of the processes in which these materials are applied and in almost every process in which they are removed. As it is nearly impossible to prevent the production and dispersal of the dust a search has been made for alternative materials not containing asbestos (7).

Hot and cold water pipes and ventilation ducts in naval ships which were previously insulated with asbestos cloth are now covered with glass fibre cloth, felt, canvas or polyurethane insulation. Sound insulating asbestos boards have been replaced by those made of glass fibre and this material has also taken the place of sprayed asbestos as an environmental insulation for steel deckheads and bulkheads.

The higher temperatures encountered in the machinery compartments have made the task of finding alternative insulating materials very difficult, and at present there do not appear to be practical alternatives to asbestos containing materials in engine rooms and boiler rooms. The material not only has to be an efficient insulator, but also has to be robust enough to withstand long periods of intense heat, vibration, and the mechanical trauma inevitable in the maintenance of machinery at sea.

Various materials are on trial, all of which are designed to reduce the amount of asbestos dust liberated during their application or removal. These include asbestos cloth treated with a dust suppressant, asbestos cloth, coated with an impermeable plastic film, and various types of glass fibre cloth. Moulded sections which are applied beneath the cloth and which previously contained 96% + amosite asbestos are now made of calcium silicate with 15% asbestos as a binder. We are experimenting with an asbestos free magnesia section overlying a calcium silicate section containing only 5% asbestos in order to see whether this is technically effective insulation, and whether or not there is a significant reduction in the asbestos dust produced in its manipulation. Instead of using asbestos rope we are using glass fibre rope. All the other minor uses of asbestos materials in which dust is produced are being surveyed and alternative materials specified.

Methods of dust suppression are largely confined to the processes in which asbestos lagging material is being applied, or where it is being processed in a workshop. Local exhaust ventilation, damping the materials, and pre-mixing or cutting of materials all helps to reduce the amount of dust released into the general working environment. For the removal of lagging materials in ships we have found damping methods increase the time taken for the work, increase the weight of the debris, and increase the difficulty of cleaning with vacuum cleaners, and for these reasons they have been abandoned.

It has been possible with suitable planning to arrange that work producing high concentrations of asbestos dust is done in isolation and that the men involved are properly protected. Table I shows the classification of the workers in the dockyards. As the result of recent dust sampling experience we have attempted to classify the main types of work according

examination and measurement of Forced vital capacity (FVC) and one second $_{1.0}$ Forced Expiratory Volume (FEV $_{1.0}$).

A survey of both "asbestos workers" and "neighbourhood workers" is nearing completion and this has also included an estimation of Gas transfer factor and its subdivisions with the purpose of finding out the most suitable methods of examination for the future supervision of these men.

Mortality studies are in progress to find out if there has been an increased mortality from lung or gastro intestinal cancer without obvious asbestosis both in the "asbestos workers" and the general working population of the dockyards.

The present indications are that while there is a fairly high incidence of asbestosis among asbestos workers, and a large number of men in the general dockyard population show changes associated with asbestos exposure, there does not seem to be a general increase in cancer mortality.

All the measures that we have taken seem to be perfectly simple and are based on the common-sense application of our present knowledge. I think that the mere fact of their existence reflects great credit on the whole-hearted efforts of both management and men, in what is a large and extremely complex industry. I hope that if the present standards are maintained, or improved, then we shall be able to see the benefit in steadily decreasing morbidity and mortality rates in fifteen to twenty years time.

TABLE I

CLASSIFICATION OF WORKERS

REGISTERED ASBESTOS WORKERS	ALL:- LAGGERS MATTRESS MAKERS SPRAYERS STRIPPERS SOME:- BOILERMAKERS CLEANERS JOINERS MASONS SMITHS STOREMEN
NEIGHBOURHOOD WORKERS	BOILERMAKERS BURNERS CAULKERS ELECTRICAL FITTERS ENGINE FITTERS JOINERS LABOURERS PAINTERS PLUMBERS SHIPWRIGHTS WELDERS ETC.

TABLE II

PROTECTIVE MEASURES FOR MOST HAZARDOUS PROCESSES

PROCESS	REMOVAL OF SPRAYED CROCIDOLITE ASBESTOS	REMOVAL OF LARGE AMOUNTS OF PIPE LAGGING
NOTICES	SPRAYED ASBESTOS REMOVAL NO ENTRY	ASBESTOS DE-LAGGING NO ENTRY
REGISTERED ASBESTOS WORKERS	IMPERVIOUS SUIT AIR FED RESPIRATOR SHOWERS	IMPERVIOUS SUIT AIR FED HOOD OR POSITIVE PRESSURE POWER RESPIRATOR SHOWERS
NEIGHBOURHOOD WORKERS	NOT ALLOWED ENTRY IF ENTRY IS VITAL, PROTECTION AS FOR REGISTERED WORKER	
SUPERVISORS	NYLON/PLASTIC SUIT. FULL FACE PIECE RESPIRATOR WITH HOOD OR POSITIVE PRESSURE POWER RESPIRATOR.	
VISITORS	VISITS TO BE LESS THAN 30 MINUTES. IF LONGER PROTECTION AS FOR REGISTERED WORKER.	

TABLE III

PROTECTIVE MEASURES FOR HAZARDOUS PROCESSES

PROCESS	APPLICATION OF LARGE AMOUNTS OF PIPE LAGGING	APPLICATION OR REMOVAL OF SMALL AMOUNTS OF PIPE LAGGING
NOTICES DISPLAYED	ASBESTOS WORK IN PROGRESS RESPIRATORS TO BE WORN	ASBESTOS WORK IN PROGRESS
REGISTERED ASBESTOS WORKERS	NYLON SUIT. APPROVED DUST RESPIRATOR SHOWERS	
NEIGHBOURHOOD WORKERS	PREFERABLY NOT ADMITTED IF ENTRY REQUIRED PROTECTION AS FOR REGISTERED WORKER	NO RESTRICTION ON ENTRY RESPIRATORS AVAILABLE
SUPERVISORS VISITORS	NO RESTRICTION ON ENTRY NYLON SUITS AND RESPIRATORS AVAILABLE	

TABLE IV

PROTECTIVE MEASURES FOR LEAST HAZARDOUS PROCESSES

PROCESS	ASBESTOS MATTRESS SHOP	ASBESTOS STORES	OTHER MINOR ASBESTOS PROCESSES
NOTICES DISPLAYED	ASBESTOS SHOP NO UNAUTHORIZED ENTRY	ASBESTOS STORE NO UNAUTHORIZED ENTRY	ASBESTOS WORK IN PROGRESS
REGISTERED ASBESTOS WORKERS	NYLON SUIT APPROVED DUST RESPIRATOR SHOWERS		
NEIGHBOURHOOD WORKERS	RESPIRATORS AVAILABLE		
SUPERVISORS VISITORS	RESPIRATORS AVAILABLE		

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